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Original Article Epidemiology of sleep and sleep disorders in The Netherlands

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ABSTRACT

Objective: There is a surging public interest in The Netherlands concerning sleep, sleep disorders and associated health. For a proper perspective, it is necessary to have reliable information on the prevalence of sleep characteristics at the national level. This study set out to assess prevalence rates and key characteristics of sleep and sleep disorders in The Netherlands.

Methods: In 2012, a nationally representative sample of 2089 individuals, aged 18–70 years, responded to a set of 48 questions, including the Holland Sleep Disorders Questionnaire, a validated questionnaire based on the International Classification of Sleep Disorders.

Results: Prevalence rates were: 32.1% for a general sleep disturbance (GSD), 43.2% for insufficient sleep, 8.2 for insomnia, 5.3% for circadian rhythm sleep disorder, 6.1% for parasomnia, 5.9% for hypersomnolence, 12.5% for restless legs disorder and limb movements during sleep, 7.1% for sleep related breathing disorder, and 12.2% for the presence of comorbidity, ie, the presence of two or more concurrent sleep disorders. In addition, sleep onset time as well as sleep duration showed U-shaped relationships with GSD prevalence rates, with respectively the 22:00–24:00 period and seven to 8 h as optimal associates. *Conclusions:* Sleep disorders and insufficient sleep have a high prevalence. As matter of concern, female adolescents reached the highest prevalence rates for most sleep disorders, insufficient sleep and daytime malfunctioning.

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1. Introduction

The annual number of papers on the epidemiology of sleep is rising rapidly [1,2]. This upsurge in scientific interest is flanked by a gradual increase in public receptivity to knowledge about the potential impact of insufficient and disturbed sleep on human error, health and disease [3–5]. These developments stress the need for valid and reliable data on the prevalence of sleep disorders, as well as answers to questions about the nature of the associations between epidemiological data and public health.

Figures on the prevalence of sleep disorders in The Netherlands are scarce and of limited validity. The most recent results were derived from health interviews with nearly 20,000 patients (aged 12 years and over) from general practices [6]. For positive responses to the single question: 'During the last two weeks, did you have

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complaints about insomnia or any other sleep disturbance?,' an overall prevalence rate of 27.3% (males 21.2% vs females 33.2%) was reported. Spoormaker and van den Bout [7] administered the validated SLEEP-50 sleep disorders questionnaire to 402 adults that were selected quasi-randomly from the 12 provinces of the Netherlands. Overall, 23.5% of the respondents were diagnosed with at least one sleep disorder (no information on gender differences). Other reports have (also) been limited by biased population samples and/or non-validated questions or questionnaires [8–10].

The present study intended to assess the prevalence of sleep disorders in a representative population sample of the Dutch population, using a validated sleep disorders questionnaire, based on the International Classification of Sleep Disorders (ICSD-2; [11]). In addition, information was collected about the habitual timing and duration of sleep. This information was deemed clinically relevant as 1. Sleep timing acts (mainly through the associated exposure to light) as a powerful synchronizer for the circadian system [12,13], impacting structure and quality of sleep [14,15], and 2. Both short and long sleep may hint at an increased risk of daytime sleepiness, fatigue, and/or ill health [4].





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2. Methods

2.1. Sample

The database of the present study consists of 2089 individuals, sampled from an ISO 26362-certified online research panel (www. motivaction.nl/en/specialties/stempuntnu) of over 80.000 citizens of The Netherlands (total population in 2012: 16.730.000). An internet panel is unlikely to give an accurate representation of the total population, however, as specific groups are liable to be either under- or overrepresented. Therefore, a widely-accepted remedy, ie, the propensity weighting technique [16] was applied to the present database, thereby correcting for standard demographic, socio-economic and/or cultural characteristics, characteristics of non-internet users, and also for non-responders. Propensity weighting scores were derived from the 'Golden Standard' defined by Statistics Netherlands (CBS). As a result, a nationally representative sample of 2089 individuals aged 18-70 years was obtained (as a consequence of the applied weighting procedure, analyses may slightly differ in the number of subjects included). In November 2012, data were collected in response to 48 questions coming from three self-report questionnaires (see assessment).

2.2. Assessment

Participants completed the following three sets of questions.

- 1. A set of seven questions about demographic (age and gender), socio-economic (education, work, income), and psychosocial (partnership and children) characteristics.
- 2. The Holland Sleep Disorders Questionnaire (HSDQ; [17]) is a clinically validated questionnaire that is composed of ICSD-based clusters of sleep complaints/symptom descriptions that are specific to six main sleep disorders, and allows the clinician to determine whether the respondent meets the diagnostic criterion/criteria for one or more of these sleep disorders. This diagnostic approach implies that subthreshold or ICSD-unspecified combinations of symptoms may pass unnoted, although they can be associated with significant distress and dysfunction. The chance of slipping through the ICSD-net of sleep disorder diagnoses is enhanced by the high prevalence of comorbidities, as also evidenced by the results of the present study (cf. Section 3.4.7). Inspired by the recent literature on transdiagnostic processes (eg, [18,19]), an attempt was made to capture not only the six distinct sleep disorders, but also the cross-cutting, comorbid aspects of disturbed sleep by calculating a General Sleep Disturbance (GSD) index, ie, the overall mean value of the 32 rating responses on the HSDQ. Thus, the GSD-index may be considered a global, 'transdiagnostic' index of disordered sleep². The clinical relevance of the GSD-index is evidenced by its more than satisfactory discriminative validity, ie, its power to distinguish clinically diagnosed patients from individuals without sleep complaints [17]. The HSDQ items are listed in Table 1, together with their factor loadings (strength of association) on the particular sleep disorder. Diagnostic accuracy [P(A) = 0.95], internal consistency (Cronbach's $\alpha = 0.90$) and overall accuracy (88%) are

satisfactory. In responding to the 32 items (rated on a five-point scale ranging from 'not at all applicable' to 'applicable'), subjects were asked to consider the past three months. Thus, scoring of the HSDQ yields 1) a mean score (GSD) that can be evaluated against a clinically validated criterion value (Cronbach's α for this study = 0.94), and 2) factor scores for six subscales, corresponding with: Insomnia ($\alpha = 0.92$), Parasomnia ($\alpha = 0.85$), Circadian Rhythm Sleep Disorder (CRSD; $\alpha = 0.84$), Hypersomnolence ($\alpha = 0.78$), Restless Legs Syndrome-/Limb Movements during Sleep (RLS/LMS; $\alpha = 0.81$), and Sleep-related Breathing Disorder (SBD; $\alpha = 0.62$). Comorbidity was scored if a participant met the criteria for two or more specific sleep disorders.Considering their special relevance for an assessment of daytime functioning, the following two HSDQ items were also analyzed as separate variables: 'During the day, I suffer from fatigue,' referred to Daytime fatigue, and 'Because of insufficient sleep, I don't function as well during the day,' referred to Daytime dysfunction (both items load on the Insomnia factor score of the HSDQ).

3. The third set consisted of nine additional sleep-related variables, as specified in Table 2. The scores for the two morningness—eveningness variables were combined into one five-point scale. In addition, the variable 'sleep deficit' was calculated by subtracting individual values of habitual sleep duration from individual values of subjective sleep need. The presence of 'insufficient sleep' was defined as a sleep deficit of 1 h or more [20].For the analyses of sleep onset times, shift workers (defined as working in rotating shifts or permanent shifts outside the traditional day shift from 9:00 a.m. to 5:00 p.m.) [21], were filtered out of the database, leaving 1779 (85.2%) cases.

2.3. Analyses

Continuous variables were analyzed by using analyses of variance (ANOVA) or analyses of covariance (ANCOVA) with covariates as mentioned in the Results section, followed by post-hoc Bonferroni-adjusted comparisons if a statistically significant main effect was present. In case of ANCOVAs, uncorrected means and standard deviations (M \pm SD) are displayed in the figures and tables. Categorical variables were analyzed by using χ^2 tests, followed by Bonferroni-adjusted pairwise z-tests. Where appropriate, point prevalence ratios (PR) and their 95% confidence intervals (95%CI) are presented. Multiple regression analyses were applied to gain insight into the relation between criterion variables (eg, sleep onset times and sleep duration), and predictor variables (age, gender, chronotype, sleep need, partnership, children at home, work, and education level). Binary logistic regression analyses were applied to determine whether this set of predictor variables (plus sleep duration and weekday sleep onset) contributed to the risk of a general sleep disturbance and six specific sleep disorders, and to compare the size of the statistically significant risk factors.

SPSS 23 for Windows was used for all statistical analyses.

3. Results & discussion

3.1. Sleep timing

Under natural conditions, the circadian system optimizes the timing of sleep to perform its restorative and adaptive functions [22,23]. However, humans in particular do not always align their time domain with the earth's rotation. Imposed (eg, shiftwork) and self-chosen (eg, weekend vs weekdays) sleep shifts cause frequent circadian mismatches, which deteriorate sleep quality and increase the risk of a wide variety of health problems [3,24]. Thus,

² Consistent with this view, a suprathreshold GSD score does not necessarily go together with suprathreshold score (-s) for one or more specific sleep disorders. That is, a general sleep disturbance can have clinical significance in the absence of a distinct sleep disorder. For the present study, this applies to 9.6% of the population sample, whereas 22.5% obtained suprathreshold scores for both the GSD-index and one or more specific sleep disorders.

Table 1

HSDQ items (and their factor loadings) for the 6 specific sleep disorders.

Specific sleep disorders and their relevant HSDQ-items
Insomnia
The quality of my sleep is poor and I don't feel well rested in the morning (0.83)
Because of insufficient sleep, I don't function as well during the day (0.83)
Especially after a bad night, I suffer from one or more of the following consequences: fatigue, sleepiness, bad mood, poor concentration, memory problems, and lack of
energy (0.81)
During the day, I suffer from fatigue (0.80)
Despite having plenty of opportunity to sleep in, I don't get enough sleep (0.75)
I worry about the consequences of my poor sleep (e.g. for my health) (0.71)
I have difficulty falling asleep at night (0.58)
At night, I lie awake for a long time (0.45)
Circadian rhythm sleep disorder
I have difficulty falling asleep at night (0.79)
I sleep poorly because I don't manage to fall asleep at a normal hour and wake up at a normal hour in the morning (0.78)
The time at which I fall asleep varies strongly from day to day (0.67)
At night, I lie awake for a long time (0.66)
l don't fall asleep until the morning and have great difficulty waking up early. I sleep in on weekends (0.62)
When I have to stay awake during the night, my daytime sleep is poor (0.36)
Parasomnia
Quite often I partially wake up and find myself thrashing my arms. I usually don't recall this later on (0.71)
I suffer from nightmares or bad dreams (0.71)
I regularly have vivid dreams in which I am being attacked and try to defend myself with uncontrolled movements (0.70)
I wake up in the middle of the night, screaming and/or heavily perspiring and feeling anxious (0.66)
I suffer from sleepwalking (0.64)
I have injured myself during sleep and had no recollection of the event afterward (0.57)
Hypersomnolence/excessive daytime sleepiness
I fall asleep repeatedly throughout the day (0.82)
During the day, I suffer from sleep attacks that are so severe that I cannot suppress them (0.79)
During the day, I fall asleep involuntarily, especially in monotonous situations (e.g. during a boring TV show) (0.78)
I usually sleep more than 10 h at night, have difficulty waking up in the morning, and nap during the day (0.53) After a daytime nap I don't feel refreshed (0.41)
Restless legs syndrome & leg movements during sleep
When I am sitting still, especially in the evenings, I feel an urge to move my legs (0.79)
When I experience 'restless legs,' I can suppress these sensations by walking or stretching my legs (0.74)
When I lie down in bed, I experience unpleasant, itchy, or burning sensations in my legs (0.72)
While asleep I suffer from kicking leg movements that I just cannot suppress (0.65)
I move my arms or legs during sleep (0.64)
Sleep related breathing disorders
I stop breathing during sleep (0.82)
I snore loudly while I am asleep (0.77)
At night I wake up with a start feeling like I am choking (0.60)
I wake up with a dry mouth in the morning (0.53)

Table 2Additional sleep-related variables (cf. Section 2.2).

Habitual sleep onset weekdays

If you work at fixed times during weekdays or are unemployed, at what time do you usually fall asleep on weekdays? Before 22:00/Between 22:00 and 23:00/Between 23:00 and 24:00/Between 24:00 and 01:00/After 01:00 Habitual sleep onset weekend

At what time do you usually fall asleep in the weekends?

Before 22:00/between 22:00 and 23:00/between 23:00 and 24:00/between 24:00 and 01:00/after 01:00

Habitual sleep duration

How many hours do you usually sleep at night? Less than 5 h/5 h/6 h/7 h/8 h/More than 8 h

Subjective sleep need

How many hours of sleep do you need on average?

Less than 5 h/5 h/6 h/7 h/8 h/More than 8 h

Sleep quality

_

I am a very poor/poor/average/good/very good sleeper.

Use of hypnotics

Do you use prescribed medication in order to promote your sleep?

Yes, daily/yes, frequently/yes, now and then/no

Frequency of napping

At how many days do you usually nap? None/1-2/3-4/5-6/daily

Morningness

Since early age I have a morningness preference. On days off I wake up at the same time as on workdays. Not at all applicable/usually not applicable/applicable at times/usually applicable/applicable

Eveningness

Since early age I have an eveningness preference. On days off I prefer to oversleep as long as possible. Not at all applicable/usually not applicable/applicable at times/usually applicable/applicable information about (deviant) sleep timing is a key factor in evaluating sleep quality. The present study selected sleep onset time as parameter of sleep timing, considering that most day workers most likely vary their sleep timing by delaying or advancing their bedtime, while– due to 9:00 a.m.–5:00 p.m. work commitments– maintaining a set wake time.

For the total population sample minus the shift workers (N = 1779; cf. Section 2.2), the frequency distributions of sleep onset times for weekdays as well as weekend days were skewed left, failed to meet the criterion of normality (Kolmogorov D = 0.201 and 0.191 respectively, both p < 0.001), and had median values of 23:00 and 24:00 and mean values of 23:02 (95%CI: 22:59–23:05) and 23:30 (95%CI: 23:27–23:33), respectively. Thus, the weekend shift (ie, the mean delay of sleep onset on weekend days vs weekdays), also referred to as 'social jet lag' [25], was 28 min (SD: 42 min).

3.1.1. Predictors

On weekdays in particular, sleep onset time is a likely outcome of a trade-off between individual biological predispositions (gender, chronotype, sleep need, etc.) and social conditions/restrictions (work schedule, family, leisure, education, etc.) [26]. In the present study, this was verified by applying multiple regression analysis, with "weekday sleep onset" as criterion variable and "age," "gender," "chronotype," "sleep need," "partnership," "children at home," "day-work vs non-work," and "education level" as predictor variables. Sleep onset times appeared significantly associated with nearly all eight predictor variables, with the exception of partnership (F (8,1770) = 66.84, p < 0.001; adjusted $R^2 = 0.23$). Ranked by decreasing value of their standardized regression coefficients β (cf. Table 3), later weekday sleep onset times appeared associated with propensity towards eveningness, non-work vs daywork, lower sleep need, advancing age, higher education level, absence of children at home, and male gender.

Multiple regression analysis for weekend days showed very similar, although somewhat reordered results (F (8,1770) = 59.72, p < 0.001; adjusted $R^2 = 0.21$), with later sleep timing associated with eveningness propensity as the strongest predictor, followed by lower sleep need, no children at home, non-work vs day-work, male gender, higher education, and no significant contributions by the predictors age and partnership (cf. Table 3). Comparing the β -weights for weekend vs weekday sleep timing, the most striking differences clarified that weekend sleep timing had a weaker association with work condition, and no significant association with age.

3.1.2. Age and gender

Controlling for chronotype score (cf. Section 3.1.3), the dependence of sleep onset time on age and gender was specified by ANCOVA's for the weekday and the weekend sleep onsets, respectively. The chronotype-corrected results showed significant overall impacts of age (F (4,2033) = 30.66, p < 0.001 and F (4,2033) = 8.11, p < 0.001, respectively) and gender (F (1,2033) = 25.22, p < 0.001 and F (1,2033) = 36.74, p < 0.001, respectively), and a significant age × gender interaction (F (4,2033) = 2.62, p < 0.05 and F (4,2033) = 3.13, p < 0.05, respectively). The (tilted) U-shaped trends in Fig. 1 demonstrate overall later onset times for males vs females, relatively late sleep onsets for both male and female adolescents and seniors, and relatively advanced sleep times for individuals in their thirties and forties. Note that the overall age-related range for females is about twice as large compared to that for men (0.52 vs 0.27 h), and that with advancing age, the gender difference diminishes and ultimately dissolves.

Aging also correlated with the magnitude of the weekend shift, as it decreased monotonically with advancing age, from 40.3 min for the youngest to 16.4 min for the oldest age category (F (4,2034) = 30.82, p < 0.001). Weekend shift size did not differ between sexes.

Table 3

Statistically significant (p < 0.05) associations between predictor variables (columns) and criterion variables (rows). A: regression coefficients β ; B: Odds ratio's with 95% confidence intervals.

	Age	Gender	M/E-ness	Sleep need	Partner	Child	Work	Education	Sleep onset	Insufficient sleep
Α										
Regression coe	fficients									
Sleep onset weekday	0.15	-0.08	0.33	-0.18		-0.10	-0.23	0.12	-	-
Sleep onset weekend		-0.08	0.38	-0.19		-0.12	-0.11	0.07	-	-
Sleep duration	-0.11	0.04		0.42			-0.05		_	_
В										
Odds ratio's										
GSD	0.98 (0.97–0.99)		1.09 (1.04–1.14)		0.79 (0.62–1.0)		0.44 (0.34–0.57)	0.91 (0.86-0.96)		3.45 (2.77-4.29)
Insomnia							0.39 (0.26–0.59)		1.46 (1.21–1.76)	6.86 (4.44-10.59)
CRSD			1.11 (1.00–1.23)				2.16 (0.91–4.10)		1.94 (1.50–2.49)	6.14 (3.50–10.77)
Parasomnia	0.95 (0.94–0.97)		(1.56 (1.01–2.41)	0.37 (0.23–0.59)			1.59 (1.05–2.39)
Hyper- somnolence	0.97 (0.96–0.99)	0.63 (0.41–0.96)					0.31 (0.19–0.50)		0.78 (0.63-0.97)	
RLS-LMS	()	()					0.63 (0.45–0.88)	0.83 (0.77-0.89)	1.17 (1.00–1.36)	2.97 (2.19-4.03)
SBD		0.46 (0.32–0.67)					0.60	0.87 (0.79-0.95)		2.01 (1.39-2.91)
Comorbidity	0.98 (0.97–0.99)	(0.32 0.07)	1.08 (1.01–1.16)				(0.35 ⁻ 0.51) 0.51 (0.36–0.72)	0.88 (0.81-0.94)		3.06 (2.24-4.19)



Fig. 1. Motivated by the great similarity of the quadratic trends for weekdays and weekend days (cf. Section 3.1.2) and the strong correlation of their sleep onset times (Pearson: 0.75, N = 1779, p < 0.001), weighted means (i.e. [(5 × weekday onset time) + (2 × weekend onset time)]/7) and standard errors were calculated and used to illustrate age- and gender-related trends.

3.1.3. Chronotype

A well-documented endogenous source of inter-individual variation in sleep timing is known as chronotype, originally dubbed morningness/eveningness [27,28]. Based on the five morningness-eveningness categories used in the present study, individuals with a score of '1' were referred to as morning-persons (6.9% of the total population sample, 95%CI: 5.9-8.1) and individuals with a score of '5' as evening-persons (10.3% of the total population sample, 95%CI: 9.1–11.7). These extreme subgroups did not differ in their proportions of females and males, but differed significantly with respect to their age (F (1,412) = 65.16, p < 0.001), ie, the subgroup of morning-persons appeared older (M \pm SD: 53.8 \pm 11.3 y) than the subgroup of evening-persons (M \pm SD: 43.0 ± 14.9 y). This group difference most likely resulted from the overall shift from relative eveningness propensity during adolescence towards relative morningness propensity during senescence. observed before [29]. For the present population sample, this finding was replicated by the negative correlation between eveningness score and age (Pearson: -0.21, N = 2089, p < 0.001).

Sleep onset times were analyzed in relation to chronotypescores by applying ANCOVA's with chronotype as main factor and age as covariate (see above). For weekdays as well as weekend days, chronotype had a significant, age-corrected effect, as disclosed by linear and quadratic trends (F (4,2038) = 64.29, p < 0.001 and F (4,2038) = 82.92, p < 0.001, respectively). The earliest sleep onsets were reported by the morning-persons (M \pm SD for weekdays 22:49 \pm 1:03; for weekend days 23:01 \pm 0:59; a weekend shift of 12 min) and the latest sleep onsets were reported by the eveningpersons (M \pm SD for weekdays 23:50 \pm 1:04; for weekend days 24:18 \pm 0:50; a weekend shift of 28 min), about 1 h later than morning-persons. Also, as apparent from these mean values for weekend vs weekday sleep onsets and evidenced by ANCOVA with chronotype as main factor and age as covariate (F (4,2038) = 4.04, p < 0.01), weekend shift size increased systematically from extreme morningness (M \pm SD: 0.21 \pm 0.54 h) towards extreme eveningness propensity (M \pm SD: 0.54 \pm 0.71 h).

3.1.4. Conclusion

Overall, sleep onset times were later for males vs females, a difference that tended to decrease with advancing age. For both genders, starting in middle age, sleep onset times systematically delayed with advancing age, a trend that appeared independent of (and opposite to) the trend from eveningness propensity in adolescence towards morningness propensity in senescence. In addition, sleep onset time proved a weighted resultant of a selection of (chrono-) biological and psychosocial factors, among which eveningness propensity and a condition of not working turned out to be the strongest predictors. Eveningness accounted for the largest amount of variance in sleep onset time, in particular during weekends. The impact of eveningness on weekend sleep also manifested itself in the size of the weekend shift for extreme evening persons, ie. more than twice the size for extreme morning persons. Therefore, recognizing that the timing of weekday sleep is less strongly influenced by eveningness propensity than the timing of weekend sleep (cf. Table 3), and that the reverse applied for work condition (viz more impact on weekdays), these results suggest that, on weekdays, evening persons are forced to dissociate from their preferred sleep timing, more so than morning-persons. Of note, this applies a fortiori for the adolescents/young adults, considering their maximal eveningness propensity.

3.2. Sleep duration

A growing number of studies converge on the observation that both short and long sleep are associated with increased risk of allcause mortality and morbidity, including obesity, diabetes and cardiovascular disease [30]. In particular short sleep (preferably assessed as 'insufficient sleep,' recognizing the existence of naturally short sleepers; see below) may serve as a proxy for sleep disturbance, as individuals who report short/insufficient sleep are also more likely to report difficulty initiating sleep, difficulty maintaining sleep, early morning awakenings, and non-restorative sleep [31]. However, subjective reports of sleep duration should be interpreted cautiously, recognizing observations of only moderate correlations with objective sleep duration measures [32], and findings of systematic overestimation by self-reported good sleepers versus underestimation by insomniacs [33–35].

For the population sample under study, the frequency distribution of the sleep duration reports was skewed left, deviated from a normal distribution (Kolmogorov–Smirnov statistic = 0.206, df = 2029, p < 0.001), and had a median value of 7 h and a mean \pm SD of 6.97 \pm 1.14 h. Overall, 67.8% (95%CI: 65.8–69.8) of the sample reported a sleep duration of 7 h or less, and 30.4% (95%CI: 28.4–32.4) reported only 6 h or less (commonly defined as short sleep [36]).

An average sleep duration of 7 h as well as a prevalence of short sleep of about 30% are consistent with most epidemiological studies of self-reported habitual sleep duration [37–40]. However, Knutson and coworkers [41] combined 24-h time diary data from eight different population samples, applied corrections for socio-demographic factors, and concluded that the prevalence of short sleep (less than 6 h) in 2006 had not changed significantly compared to the prevalence rate of 15%, measured 31 years earlier (although it had increased significantly among full-time workers only). In addition to socio-demographic factors, methodological differences (diary measurements vs subjective reports) might have led to this deviating estimate of the prevalence of short sleep.

3.2.1. Predictors

Unfortunately, the current data set did not allow separate regression analyses for weekend vs weekday sleep duration. Overall, sleep duration appeared most strongly associated with sleep need. Age, work and gender were the only other significant predictor variables (cf. Table 3) (F (8,2020) = 70.29, p < 0.001; adjusted $R^2 = 0.22$).

3.2.2. Age and gender

The impact of age and gender on sleep duration was specified by ANCOVA's, controlling for the significant predictor variables 'sleep need' and 'work'. The corrected results showed a significant effect of age (F (4,2020) = 10.94, p < 0.001), a borderline effect of gender (F (1,2020) = 3.69, p = 0.05), and a significant age \times gender interaction (F (4,2020) = 9.74, p < 0.001). As shown in Fig. 2, mean sleep duration peaked in adolescence, for both females (7.79 h, 95%

CI: 7.63–7.95) and males (7.26 h, 95%CI: 7.00–7.52), and linearly decreased across successive age categories (with the male seniors as 'outliers') (F (4,2307) = 23.48, p < 0.001). Overall, females reported 0.33 h more sleep than males (F (1,2307) = 61.22, p < 0.001). The age-related decrease as well as the higher females-related values of sleep duration are consistent with previous studies [42].

3.2.3. Sleep need

Consistent with their longer sleep, females reported a higher sleep need than males (cf. Fig. 2), with a mean gender difference of 0.53 h, as verified by ANCOVA with gender and age as main effects and sleep duration as covariate (F (1,2021) = 56.92, p < 0.001). Moreover, sleep need diminished significantly across age categories (F (4,2021) = 12.26, p < 0.001), although for the first three age categories females showed stable values, that only began to decline after the 35–44 age category. The overall correlation between sleep need and sleep duration was statistically significant and did not differ between males and females (Pearson: 0.45, N = 2029, p < 0.001). The frequency distribution of sleep need was skewed left, deviated from a normal distribution (Kolmogorov-Smirnov statistic = 0.273, df = 2089, p < 0.001), and had a median value of 8 h and a mean \pm SD value of 7.51 \pm 0.92 h, ie, almost 1 h more than the mean sleep duration.

3.2.4. Insufficient sleep

The grand mean (±SD) value of sleep deficit (sleep need minus sleep duration; cf. Section 2.2) was 0.52 ± 1.10 h, with a significant variation across age (F (4,2022) = 8.19, p < 0.001) and a significant age × gender interaction (F (4,2022) = 7.16, p < 0.001). Fig. 2 clarifies that sleep deficit was prominent in the three middle age categories and much less in the youngest and (only for males) oldest groups. Overall, sleep deficit correlated negatively with sleep duration (Pearson: -0.66, N = 2029, p < 0.001).

The overall prevalence of insufficient sleep (sleep deficit ≥ 1 h; cf. Section 2.2) was 43.2% (95%CI: 41.1–45.3), and higher for females (45.9%, 95%CI: 42.9–48.8) than for males (40.2%, 95%CI: 37.2–43.3) (z-test with Bonferroni correction p < 0.05). For both genders, the prevalence of insufficient sleep was maximal during midlife (ie, 35–45 years). The overall prevalence rate is approximately twice that reported by Hublin and co-authors [20] and Ursin and co-authors [43], who reported prevalence rates of respectively 20.4% and 20%. Both studies, however, analyzed data that were collected considerably earlier than the data of the present study (end 2012), viz 1981/1990 and 1997–1999. Considering reports of a growing (doubling) prevalence of sleep deficiency [40,44], this trend might account for (part of) the difference in prevalence estimates.

Insufficient sleepers (ie, those who met this definition) vs 'sufficient' sleepers judged themselves as worse sleepers (F



3.2.5. Conclusion

Sleep duration appeared to be associated mainly with sleep need, with modest roles for the predictors age, work and gender. Both sleep duration and sleep need reached higher levels for females than for males, and tended to decrease with advancing age. Most importantly, the prevalence rate of insufficient sleep (43.2%) was high, higher for females than for males, reached maximal values during midlife and was associated with daytime fatigue and malfunctioning.

3.3. General sleep disturbance

The point prevalence of a general sleep disturbance (cf. Section 2.2) was estimated at 32.1% (95%CI: 30.2–34.2). Fig. 3 and Table 4 present prevalence rates broken down by gender and age category. A logistic regression analysis was applied to determine whether the previously introduced predictor variables (plus weekday sleep onset, and insufficient sleep in place of sleep need) contributed to the risk of a general sleep disturbance. Significant odds ratio's (OR; cf. Table 3) testified that higher odds of a general sleep disturbance were associated with higher levels of insufficient sleep and eveningness propensity, whereas lower odds were



Fig. 3. Prevalence ratio's (with upper 95% confidence intervals) of a general sleep disturbance for males and females vs age categories.



Fig. 2. Means (with standard errors) of sleep duration and sleep need, calculated separately for males and females as a function of age category. Sleep need minus sleep duration is designated as sleep deficit.

Table 4

Sleep disorders	PR	95%CI	M/F	Age categories $18-24 \rightarrow 55-70$
General sleep disturbance	32.1	30.2-34.2	M 29.2	31.3 - 33.1 - 32.6 - 31.8 - 21.6
			F 34.8	52.7 - 27.3 - 32.5 - 30.6 - 35.3
Insufficient sleep	43.2	41.1-45.3	M 40.2	39.3 - 38.5 - 48.3 - 50.7 - 27.4
			F 45.9	30.9 - 48.0 - 53.3 - 47.4 - 45.1
Insomnia	8.2	7.0–9.4	M 6.8	3.6 - 6.5 - 8.9 - 8.5 - 4.8
			F 9.5	10.9 - 6.4 - 13.3 - 7.3 - 9.4
CRSD	5.3	4.4-6.3	M 4.7	0.0 - 4.1 - 9.8 - 5.2 - 2.1
			F 5.7	8.5 - 4.9 - 5.0 - 4.8 - 6.3
Parasomnia	6.1	5.2-7.2	M 6.4	7.1 - 9.4 - 10.6 - 5.7 - 1.7
			F 5.9	14.5 - 5.4 - 5.8 - 3.0 - 3.5
Hypersomnolence	5.9	5.0-7.0	M 6.5	7.1 - 7.1 - 8.5 - 5.2 - 5.1
			F 5.5	15.2 - 5.9 - 3.7 - 2.2 - 3.5
RLS-/LMS	12.5	11.2-13.9	M 12.6	19.0 - 13.6 - 14.0 - 12.8 - 8.9
			F 12.4	15.2 - 8.8 - 8.7 - 12.1 - 17.3
SBD	7.1	6.1-8.3	M 8.8	9.6 - 7.1 - 11.5 - 9.0 - 7.2
			F 5.6	6.7 - 3.4 - 6.2 - 4.8 - 6.7

Prevalence rates (PR) and 95% confidence intervals (95%CI) for the specified sleep disorders. In addition, PR's were calculated separately for males (M) and females (F), and the 5 age categories (18–24, 25–34, 35–44, 45–54, 55–70 years). All figures in percentages.

associated with regular day-work (vs non-work and shift-work), partnership, higher education level, and advancing age.

3.3.1. Gender and age

Overall, GSD prevalence appeared significantly higher for females (PR 34.8%, 95%CI: 32.1–37.7) than for males (PR 29.1%, 95%CI: 26.4–32.0) ($\chi^2 = 7.73$, df = 1, p < 0.01). Most notably, the categories of adolescents and seniors stand out. As shown in Fig. 3 (and Table 4, GSD prevalence among female adolescents (52.7%, 95%CI 45.1–60.2) was significantly higher than among their male peers (31.3%, 95%CI 22.4–41.9) (z-test with Bonferroni correction p < 0.05). For the senior category, the gender difference tended to significance (p < 0.10). The marked GSD prevalence for female adolescents (and seniors) appeared to be a major factor underlying the significant overall impact of age ($\chi^2 = 26.78$, df = 4, p < 0.001).

Potential daytime correlates of the occurrence of GSD were evaluated by applying ANOVA's with Gender and Age as main factors and fatigue and daytime malfunctioning as dependent variables. For both variables, gender (fatigue: F (1,2373) = 52.67, p < 0.001; malfunctioning: F (1,2373) = 13.32, p < 0.001) as well as age (fatigue: F (4,2373) = 18.19, p < 0.001; malfunctioning: F (4,2373) = 18.19, p < 0.001; malfunctioning: F (4,2373) = 13.70, p < 0.001) were statistically significant, whereas Gender × Age interactions were not (cf. Fig. 4).

These results corroborate and extend earlier reports of higher prevalence rates of sleep disturbances and daytime malfunctioning in female adolescents compared to their male peers [45]. In a sample of 1713 Swedish school students (16–18 years), Danielsson and colleagues [46] investigated longitudinally, from 2006 to 2008,



Fig. 4. Mean ratings (5-point rating scale) with standard errors for the HSDQ item 'During the day, I suffer from fatigue'.

whether catastrophic worry mediated the relationship between adolescent sleep disturbances and depressive symptoms. Their results revealed that 1. Girls reported more sleep disturbances, depressive symptoms, and catastrophic worry relative to boys, 2. Adolescents experiencing sleep disturbance tended to report concurrent catastrophic worry and depressive symptoms one year later, and 3. Catastrophic worry was a mediator of this relationship. In an impressively large group of Finnish adolescents, Kronholm and co-authors [44] investigated long-term (from 1984 to 2011) trends in insomnia symptoms, fatigue and school performance. Results showed that insomnia symptoms and fatigue were associated with lower school performance and were more prevalent among girls (11.9 and 18.4%) compared to boys (6.9 and 9.0%, respectively) (cf. [47–49]).

The present result of maximal levels of fatigue in adolescent/ early adulthood females fits in with the results of studies of chronic fatigue and chronic fatigue syndrome. In a nationally representative survey of 10,123 adolescents, Lamers and co-authors [50] showed that the prevalence of extreme fatigue - together with at least one associated symptom (pains, dizziness, headache, sleep disturbance, inability to relax, irritability) that did not resolve by resting or relaxing and lasting at least three months – was markedly higher in adolescent girls, with a girl-to-boy ratio of 3.0:1 for extreme fatigue only, and 4.3:1 for extreme fatigue with comorbid depression or anxiety, and was even higher in older adolescents. In a sample of 3467 adolescents (1718 boys and 1749 girls), ter Wolbeek and colleagues [51] observed that 9.6% of the girls and 2.3% of the boys felt severely fatigued for at least three months. In addition, multiple regression analysis with 21 lifestyle characteristics as predictor variables revealed that only sleep characteristics and the participation in sports played a role in predicting fatigue. In a follow-up study in 633 females, ter Wolbeek et al. [52] focused on the course of fatigue and its predictors during the transition from adolescence to young adulthood. The authors showed that shorter nighttime sleep was related to a rise in fatigue severity, depressive symptoms, anxiety, and symptoms of chronic fatigue syndrome.

3.3.2. Sleep timing and chronotype

Sleep timing c.q. weekday sleep onset time, showed a significant, U-shaped relationship with GSD prevalence ($\chi^2 = 70.12$, df = 4, p < 0.001), as illustrated in Fig. 5A. Apparently, the lowest prevalence of a general sleep disturbance (24.4%) is associated with a sleep onset period from 11:00 p.m. until midnight, whereas both the earliest (before 22:00) and the latest (after 01:00) sleep onset periods were associated with the highest prevalence rates (45.7% and 54.6%, respectively).



Fig. 5. Prevalence ratio's (with upper 95% confidence intervals) of a General Sleep Disturbance vs sleep timing (A) and sleep duration (B).

In order to estimate if chronotype could have impacted this result, an ANCOVA was run on the GSD score, with weekday sleep onset time as main factor and chronotype score as covariate. Even after this correction for the impact of chronotype, the significance of the U-shaped association was maintained, as evidenced by a quadratic trend (F (5,2038) = 30.33, p < 0.001). A similar ANCOVA, this time with chronotype as main factor and weekday sleep onset as covariate, confirmed the impact of chronotype, apart from the impact of sleep timing (F (4,2038) = 12.34, p < 0.001; with significant linear trend). Thus, it appeared that the U-shaped relationship between sleep timing and GSD prevalence is not dependent on the linear increase of GSD prevalence across the trajectory from extreme morningness (with a GSD prevalence of 21.5%, 95%CI: 15.6-28.9) towards extreme eveningness propensity (with a GSD prevalence of 44.2%, 95%CI: 37.7–50.9) $(\chi^2 = 45.15, df = 4,$ p < 0.001).

3.3.3. Insufficient sleep

As shown in Table 4, insufficient sleep was highly prevalent and significantly different between males and females ($\chi^2 = 39.54$, df = 4, p < 0.001) as well as between age categories for females ($\chi^2 = 21.24$, df = 4, p < 0.001) and males ($\chi^2 = 36.29$, df = 4, p < 0.001). Insufficient sleep was also associated with GSD prevalence, i.e. 45.6% (95%CI: 42.4–48.8) of the insufficient sleepers met the criterion of a general sleep disturbance.

3.3.4. Sleep duration and daytime functioning

Fig. 5B shows another U-shaped relationship, this time between sleep duration and GSD prevalence. Sleep durations less than 5 h were associated with a GSD prevalence of 80%, whereas sleep durations of seven to 8 h were associated with prevalence rates of 25%, and sleep durations of more than 8 h with a prevalence of 39.1%.

The potential impact of a general sleep disturbance is suggested by substantial correlations between the GSD score and daytime dysfunctioning as well as daytime fatigue (Pearson: 0.73 and 0.62, respectively; both N = 2089, p < 0.001).

3.3.5. Partner, work and education

Partnership appeared to 'protect' somewhat against a general sleep disturbance. Among individuals with a steady partner, GSD prevalence was 29.0% (95%CI: 26.8–31.4), whereas for singles a prevalence of 40.3% (95%CI: 36.4–44.4) was observed (χ^2 24.31, df = 1, p < 0.001).

Working in regular day-work vs shiftwork and non-work entailed a lower risk: the GSD prevalence for day-workers was 25.2% (95%CI: 22.7–27.9) versus 38.7% (95%CI: 33.5–44.2) for shiftworkers and 39.8% (95%CI: 36.2–43.5) for non-workers (χ^2 28.54, df = 1, p < 0.001).

Level of education was inversely related to GSD prevalence (χ^2 28.41, df = 7, p < 0.001): the highest level of education was associated with the lowest prevalence (PR 21.8%, 95%CI: 16.7–28.0), whereas the lowest level of education combined with the highest GSD prevalence (PR 38.4%, 95%CI: 31.9–45.3).

3.3.6. Hypnotics

Overall, 12.3% (95%Cl 11.0–13.8) of all individuals reported the use of some means to facilitate their sleep: occasionally (6.5%), regularly (2.1%) or daily (3.7%). About half of them (6.5% of the total sample, 95%Cl 5.5–7.7; females 4.1%, males 2.4%) used hypnotics on prescription, about a quarter (3.5% of the total sample, 95%Cl 2.8–4.4) reported the use of melatonin, and the remainder specified alternatives such as homeopathic remedies, alcohol, or relaxation.

Of note, about one-third of the hypnotic-users (33.8%) did not meet the GSD criterion.

3.3.7. Conclusion

The overall point prevalence of a general sleep disturbance in the Netherlands is 32.1%. Relatively high levels of insufficient sleep and eveningness propensity were identified as significant risk factors, whereas regular day-work was identified as main 'protective' factor. GSD prevalence rates showed U-shaped relationships with sleep onset time and sleep duration, with respectively the 22:00–24:00 period and seven to 8 h as optimal associates. A GSD prevalence of 52.7% was observed for adolescent females, considerably larger than the 31% for their male peers. In the same vein, adolescent females scored higher ratings for daytime fatigue and malfunctioning.

3.4. Specific sleep disorders

Table 4 presents the observed prevalence rates of GSD, insufficient sleep, and the six specific HSDQ-based sleep disorder diagnoses. Note that the sum of the prevalence rates of the six specific diagnoses (45.1%), exceeds the prevalence rate of GSD. This follows from the considerable comorbidity of specific disorders, as specified in Section 3.4.7.

3.4.1. Insomnia

The insomnia diagnosis, including complaints about poor sleep quality, daytime fatigue, malfunctioning and worries about health consequences (cf. Table 1), reached an overall prevalence of 8.2% (95%CI 7.0–9.4), and was significantly higher in females (PR 9.5%, 95%CI: 7.9–11.4) than in males (PR 6.8%, 95%CI: 5.3–8.4) ($\chi^2 = 5.54$, df = 1, p < 0.05). Studies that, like the present study, applied the strict diagnostic criteria from the ICSD-2 or DSM-IV have reported similar prevalence rates, viz in the range 6%–10% (eg, [53,54]).

Overall, there appears to be an inverse relationship between the observed prevalence rate of insomnia and the level of stringency of the inclusion criteria applied [55,56]. Similar relationships apply to other sleep disorders, eg, OSA with vs without the symptom of daytime sleepiness (cf. Section 3.4.6).

The gender effect in the present data is also consistent with previous research [45]. Logistic regression analysis (cf. Table 3) showed that higher odds of insomnia were significantly (p < 0.05 or smaller) associated with insufficient sleep and late sleep onset, whereas lower odds were associated with fixed day-work.

In regards to sleep timing, insomniacs fell asleep significantly later than the group without a sleep disturbance ('GSD-negatives') (F (1,1581) = 52.69, p < 0.001), with mean values of 23:35 vs 23:00, respectively. Sleep itself was significantly shorter for the insomniacs than for the GSD-negatives (F (1,1782) = 187.93, p < 0.001), on average 5.98 vs 7.10 h. Moreover, 79.5% of the insomniacs vs 34.6% of the GSD-negatives had insufficient sleep (χ^2 128.81, df = 1, p < 0.001).

Reports about daytime fatigue and malfunctioning confirmed that insomniacs vs GSD-negatives obtained significantly higher scores (F (1,1819) = 668.01, p < 0.001 and F (1,1819) = 1803.36, p < 0.001, respectively).

3.4.2. Circadian rhythm sleep disorder

The CRSD cluster is characterized by symptoms of insomnia and sleepiness, resulting from a mismatch between the desired timing of sleep and the circadian rhythm in sleep propensity (cf. Table 1). In determining the prevalence of CRSD, it is necessary to distinguish the various types of CRSD, as they may differ widely in their ontogeny, symptomatology and specificity. Types of CRSD have been classified as extrinsic (shift-work and jetlag disorders) versus intrinsic, i.e. a dysfunction of the circadian clock or its afferent and efferent pathways (delayed sleep phase, advanced sleep phase, irregular sleep-wake rhythm, and non-24-hour, so-called free-running disorders). Prevalence rates reported for these types of CRSD (e.g. [57] range from less than 1% for advanced and delayed sleep phase disorder to 10% for shift-work disorder [58].

For the present study, the overall prevalence of CRSD was estimated at 5.3% (95%CI: 4.4 - 6.3) (cf. Table 4). Logistic regression analysis revealed that higher odds of CRSD were significantly associated with insufficient sleep, shift-working, later sleep onset time and stronger eveningness propensity (cf. Table 3). Sleep onset time was significantly later for the CRSD-positives than for the GSD-negative group (mean values 24:01 vs 23:00) (F(1,1509) =96.03, p < 0.001), while mean sleep duration was more than 1 h less for the CRSD-positives vs the GSD-negatives (mean values 6.00 vs 7.10 hours; F(1,1705) = 115.12, p < 0.001). Also, 73.6% of the CRSDpositives vs 34.6= of the GSD-negatives met the criterion of insufficient sleep (X^2 66.33, df = 1, p < 0.001). With respect to reports about daytime fatigue and daytime malfunctioning, CRSDpositives obtained significantly higher scores than the GSD-negatives (F(1,1749) = 319.39, p < 0.001 and F(1,1749) = 887.36, p < 0.001, respectively).

3.4.3. Parasomnia

The present study estimates the prevalence of NREM-related arousal disorders, including sleep terrors, nightmares, sleepwalking and confusional arousals, at 6.1% (95%CI: 5.2–7.2) (cf. Table 4). A telephone survey of nearly 5000 adults found a prevalence of 2.2% for night terrors, 2.0% for sleepwalking, and 4.2% for confusional arousals [59]. Several reports of the prevalence of nightmares in the general population showed percentages ranging between 3% and 8% [60], with prevalence estimates varying with the specific criteria used.

Logistic regression analysis showed that higher odds of parasomnia were significantly associated with insufficient sleep and children at home, whereas lower odds were associated with fixed day-work and advancing age (cf. Table 3).

For the parasomniacs mean sleep duration was significantly shorter than for the GSD-negatives (6.48 vs 7.10 h) (F (1,1722) = 42.53, p < 0.001), and the prevalence of insufficient sleep was higher for the parasomniacs than for the GSD-negatives (51.6% vs 34.6%; χ^2 66.33, df = 1, p < 0.001). Sleep onset times did not differ between the two groups.

Parasomniacs vs GSD-negatives obtained significantly higher scores for both fatigue (F (1,1763) = 186.31, p < 0.001) and daytime malfunctioning (F (1,1763) = 610.75, p < 0.001).

3.4.4. Hypersomnolence

Hypersomnolence, respectively excessive daytime sleepiness, is the key manifestation of a group of disorders including narcolepsy and various forms of hypersomnia, is common with obstructive sleep apnea and is also associated with a wide range of diseases, including psychiatric and neurological disorders, pulmonary and cardiac conditions [61]. Swanson and colleagues [62] estimated the prevalence of hypersomnolence at 18% and Walsleben and colleagues [63] reported a prevalence of as much as 23.9%, both using the Epworth Sleepiness Scale (ESS) score \geq 10 as cutoff. Referring to the findings from a 1997 Gallup Poll, the latter authors pointed out that 6% of the respondents reached an ESS score of \geq 15, clearly in the pathologic range. This prevalence rate fits in well with present study's prevalence of 5.9% (95%CI: 5.0–7.0) (cf. Table 4).

Logistic regression analysis showed that lower odds were significantly associated with fixed day-work, male gender, later sleep onset, and advancing age (cf. Table 3).

Sleep duration of hypersomnolence-positives was shorter as compared with GSD-negatives (mean values 6.80 vs 7:10 h; F (1,1729) = 9.96, p < 0.01). Also, the prevalence of insufficient sleep was higher for hypersomnolence-positives vs GSD-negatives (52.0% vs 34.6%) (χ^2 14.88, df = 1, p < 0.001). Sleep onset times did not differ between the two groups.

Compared with the GSD-negatives, hypersomnolence-positives reported significantly higher levels of daytime fatigue and daytime malfunctioning (F (1,1765) = 284.25, p < 0.001 and F (1,1765) = 632.35, p < 0.001, respectively).

3.4.5. Restless legs syndrome – limb movements during sleep

The HSDQ includes the diagnostic criteria for RLS as formulated by the ICSD-2 as well as by the RLS diagnosis and epidemiology workshop at NIH [64]. In addition, two more items describing limb movements occurring during sleep (LMS, without information on the periodicity of the movements) loaded on the RLS-LMS factor of the HSDQ. These items were included in an attempt to capture some of the symptoms of periodic leg movements during sleep (PLMS), commonly observed in at least 80% of RLS cases but not specific to RLS [65,66]. Moreover, the presence of PLMS is considered supportive of the diagnosis of RLS [64].

The prevalence of RLS-LMS in the present study was 12.5% (95% CI: 11.2–13.9; cf. Table 4), viz (above) the upper limit of the RLS prevalence rates reported by most studies [11,64,67,68].

Hypothetically, adding the LMS-items very likely has expanded the RLS diagnosis, and thus contributed to the high RLS-LMS prevalence. It cannot be excluded that high LMS-ratings might have been given by individuals experiencing normal sleep-related motor behavior, such as hypnic jerks/sleep starts or leg cramps [69].

Logistic regression analysis showed that higher odds of RLS-LMS were significantly associated with insufficient sleep and later sleep onset, whereas lower odds were associated with fixed day-work and higher education level (cf. Table 3).

Sleep onset was significantly later for the RLS-LMS cases than for the GSD-negatives (respective mean values 23:20 vs 23:00) (F (1,1659) = 22.46, p < 0.001). Sleep duration for RLS-LMS cases was shorter than for the GSD-negatives (mean values 6.40 vs 7.10 h, F (1,1878) = 101.38, p < 0.001). Moreover, insufficient sleep was observed in 64.4% of the RLS-LMS cases, nearly double the percentage for the GSD-negatives (34.6%) (χ^2 81.77, df = 1, p < 0.001). Also, RLS-LMS cases obtained higher scores for daytime fatigue (F (1,1921) = 369.54, p < 0.001) and daytime malfunctioning (F (1,1921) = 839.99, p < 0.001).

3.4.6. Sleep related breathing disorders

Difficulty or cessation of breathing during sleep can occur due to obstruction of the upper airway (obstructive sleep apnea, OSA), loss of ventilatory effort (central sleep apnea), or a combination. Patients with predominantly central sleep apnea constitute about 4% of the apneic individuals diagnosed in a sleep disorders center [70,71]. Thus, the large majority of apneic individuals can be expected to suffer from OSA. General population-based studies found that the prevalence of OSA (defined as \geq 5 apneas/h, and a report of daytime sleepiness) ranged from 3 to 8% in adult men and from 2% to 3% in adult women. If daytime sleepiness was omitted from the inclusion criteria (as in the current study), much higher prevalence rates were observed, ranging from 8 to 28% in adult men and 3%–26% in adult women [72]. The present results showed a SBD prevalence of 7.1% (95%CI: 6.1–8.3), 8.8% in men and 5.6% in women (cf. Table 4).

Logistic regression analysis showed that higher odds of SBD were significantly associated with insufficient sleep, whereas lower odds were significantly associated with female gender, fixed daywork, and higher education level (cf. Table 3).

Sleep onset was significantly but moderately later for the SBD-positives than for the GSD-negative group (respective mean values 23:11 vs 23:00) (F (1,1563) = 4.38, p < 0.05). Sleep was shorter for SBD-positives than for GSD-negatives (mean values 6.37 vs 7.10 h, F (1,1760) = 73.75, p < 0.001). Moreover, 58.8% of the SBD-positives vs 34.6% of the GSD-negatives fulfilled the criterion of insufficient sleep (χ^2 33.59, df = 1, p < 0.001).

Scores for daytime fatigue [F (1,1798) = 282.29, p < 0.001] and daytime malfunctioning [F (1,1798) = 630.31, p < 0.001] were significantly higher for the SBD-positives than for the GSD-negatives, confirming the daytime impact of SBD.

3.4.7. Comorbidity

10.3% of the total population sample met the criterion for one specific sleep disorder, and 12.2% was diagnosed with two or more comorbid sleep disorders. An additional 9.6% of the population sample met the criterion for a general sleep disturbance in the absence of a specific sleep disorder. Among those with comorbidity, the frequencies of the specific sleep disorders involved were: RLS-LMS (68.2%), Insomnia (53.4%), SBD (47.4%), Parasomnia (41.5%), CRSD (38.8%), and Hypersomnia (36.2%).

Logistic regression analysis showed that higher odds of comorbidity were significantly associated with insufficient sleep (OR 3.06, 95%CI: 2.24–4.19) and higher eveningness propensity, whereas lower odds were associated with fixed day-work, higher education level, and advanced age (cf. Table 3). ANOVAs with number of comorbid sleep disorders as main factor, revealed a negative linear relationship with sleep duration (F (6,2310) = 23.43, p < 0.001), and underscored the severity of comorbidity by showing positive linear relationships with the amount of sleep deficit (F (6,2310) = 43.13, p < 0.001) as well as fatigue (F (6,2376) = 102.93, p < 0.001) and daytime malfunctioning (F (6,2376) = 206.06, p < 0.001).

3.4.8. Conclusion

The prevalence of specific sleep disorder diagnoses ranged from 5.3% to 12.5% and varied substantially across age categories. On average, females reached the highest prevalence at a young age

(18–24 y), whereas males reached maximal prevalence rates in their middle age (35–44 y). Insufficient sleep, short sleep, daytime fatigue and malfunctioning distinguished all specific sleep disorders from the cases without any specific sleep disorder. Regular day-work (vs shift-work and non-work) proved the major, most consistent 'protective' factor. Comorbidity, ie, meeting the diagnostic criteria for two or more specific sleep disorders, was quite common and was observed in 12.2% of the population sample.

4. General conclusion

The point prevalence of a general sleep disturbance in a representative sample of the population of The Netherlands was calculated as 32.1%, while the prevalence rates of the six major specific sleep disorders varied between 5.3% and 12.5%. Insufficient sleep and, secondly, eveningness propensity/late sleep onset were identified as major risk factors, whereas regular day-work was identified as main 'protective' factor. The gender-related difference in the prevalence of a general sleep disturbance was largest in adolescence, where females reached a worryingly high prevalence rate of 52.7% (vs 31.3% for males). Similar gender differences were observed for the adolescence prevalence data for insomnia, circadian sleep-wake disorders, parasomnia and hypersomnolence, viz sleep disorders for which a strong association with daytime functioning was observed. Adolescence was shown to be a period of relatively late sleep timing, associated with eveningness propensity, irregular sleep-wake timing (including a relatively large weekend shift), relatively high sleep need and short sleep duration. Both sleep duration and sleep need reached higher levels in females than in males, and tended to decrease with advancing age. Yet, the difference between these two sleep variables (ie, insufficient sleep), was larger for females than for males, was maximal during midlife and was associated with daytime fatigue and dysfunction. Overall, sleep timing as well as sleep duration showed U-shaped relationships with the prevalence of a general sleep disturbance.

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Conflict of interest

None.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2016.09.015.

References

- Ferrie JE, Kumari M, Salo P, et al. Sleep epidemiology—a rapidly growing field. Int J Epidemiol 2011;40:1431–7.
- [2] Partinen M. History of epidemiological research in sleep medicine. In: Chokroverty S, Billiary M, editors. Sleep medicine. New York: Springer; 2015. p. 191–7.
- [3] Cappuccio F, Miller MA, Lockley SW. Sleep, health, and society: the contribution of epidemiology. In: Cappuccio F, Miller MA, Lockley SW, editors. Sleep, health, and society: from aetiology to public health. Oxford: Oxford University Press; 2010. p. 1–8.
- [4] Grandner MA. Addressing sleep disturbances: an opportunity to prevent cardiometabolic disease? Int Rev Psychiatry 2014;26:155–76.
- [5] Van Dongen HP, Kerkhof GA. Human sleep and cognition part II-clinical and applied research. Pref Prog Brain Res 2011;190:ix-xii.
- [6] Nielen M, Spronk I, Davids R, et al. Incidentie en prevalentie van gezondheidsproblemen in de Nederlandse huisartsenpraktijk in 2012. Uit: NIVEL Zorgregistraties eerste lijn; 2014.
- [7] Spoormaker VI, van den Bout J. The prevalence of sleep disorders; relations with depression and anxiety – a pilot study. Sleep Wake Res Neth 2005;16: 155–8.

- [8] Neven AK, Middelkoop H, Kemp B, et al. The prevalence of clinically significant sleep apnoea syndrome in The Netherlands. Thorax 1998;53:638–42.
- [9] Rijsman R, Knuistingh Neven A, Graffelman W, et al. Epidemiology of restless legs in The Netherlands. Eur J Neurol 2004;11:607–11.
- [10] Westert G, Cardol M. Slapeloosheid becijferd. Huisarts Wet 2005;48:669-70.
- [11] AASM. International classification of sleep disorders. In: Diagnostic and coding manual. 2nd ed. Westchester, Illinois: American Academy of Sleep Medicine; 2005.
- [12] Burgess HJ, Eastman CI. Early versus late bedtimes phase shift the human dim light melatonin rhythm despite a fixed morning lights on time. Neurosci Lett 2004;356:115–8.
- [13] Burgess HJ, Eastman CI. A late wake time phase delays the human dim light melatonin rhythm. Neurosci Lett 2006;395:191–5.
- [14] Dijk D-J, Archer SN. Circadian and homeostatic regulation of human sleep and cognitive performance and its modulation by PERIOD3. Sleep Med Clin 2009;4:111–25.
- [15] Kerkhof GA. Differences between morning-types and evening-types in the dynamics of EEG slow wave activity during night sleep. Electroencephalogr Clin Neurophysiol 1991;78:197–202.
- [16] Lohr SL. Sampling, design and analysis. Boston USA: Brooks Cole; 2010.
- [17] Kerkhof GA, Geuke ME, Brouwer A, et al. Holland sleep disorders questionnaire: a new sleep disorders questionnaire based on the international classification of sleep disorders-2. J Sleep Res 2013;22:104–7.
- [18] Harvey AG, Murray G, Chandler RA, et al. Sleep disturbance as transdiagnostic: consideration of neurobiological mechanisms. Clin Psychol Rev 2011;31: 225–35.
- [19] Krueger RF, Eaton NR. Transdiagnostic factors of mental disorders. World Psychiatry 2015;14:27–9.
- [20] Hublin C, Kaprio J, Partinen M, et al. Insufficient sleep—a population-based study in adults. Sleep 2001;24:392–400.
- [21] Costa G. Shift work and occupational medicine: an overview. Occup Med 2003;53:83-8.
- [22] Borbély AA, Daan S, Wirz-Justice A, et al. The two-process model of sleep regulation: a reappraisal. J Sleep Res 2016;25:131–43.
- [23] Hastings MH, Reddy AB, Maywood ES. A clockwork web: circadian timing in brain and periphery, in health and disease. Nat Rev Neurosci 2003;4:649–61.
- [24] Archer SN, Oster H. How sleep and wakefulness influence circadian rhythmicity: effects of insufficient and mistimed sleep on the animal and human transcriptome. J Sleep Res 2015;24:476–93.
- [25] Wittmann M, Dinich J, Merrow M, et al. Social jetlag: misalignment of biological and social time. Chronobiol Int 2006;23:497–509.
- [26] Asgeirsdottir TL, Zoega G. On the economics of sleeping. Mind Soc 2011;10: 149–64.
- [27] Kerkhof GA. Inter-individual differences in the human circadian system: a review. Biol Psychol 1985;20:83–112.
- [28] Roenneberg T, Kuehnle T, Juda M, et al. Epidemiology of the human circadian clock. Sleep Med Rev 2007;11:429–38.
- [29] Roenneberg T, Kuehnle T, Pramstaller PP, et al. A marker for the end of adolescence. Curr Biol 2004;14:R1038–9.
- [30] Marshall NS, Stranges S. Sleep duration: risk factor or risk marker for illhealth? In: Cappuccio F, Miller MA, Lockley SW, editors. Sleep, health, and society: from aetiology to public health. Oxford: Oxford University Press; 2010. p. 35–49.
- [31] Grandner MA, Kripke DF. Self-reported sleep complaints with long and short sleep: a nationally representative sample. Psychosom Med 2004;66:239.
- [32] Arora T, Broglia E, Pushpakumar D, et al. An investigation into the strength of the association and agreement levels between subjective and objective sleep duration in adolescents. PloS One 2013;8:e72406.
- [33] Altman NG, Izci-Balserak B, Schopfer E, et al. Sleep duration versus sleep insufficiency as predictors of cardiometabolic health outcomes. Sleep Med 2012;13:1261–70.
- [34] Lauderdale DS, Knutson KL, Yan LL, et al. Objectively measured sleep characteristics among early-middle-aged adults: the CARDIA study. Am J Epidemiol 2006;164:5–16.
- [35] Fernandez-Mendoza J, Calhoun SL, Bixler EO, et al. Sleep misperception and chronic insomnia in the general population: the role of objective sleep duration and psychological profiles. Psychosom Med 2011;73:88.
- [36] Grandner MA, Patel NP, Gehrman PR, et al. Problems associated with short sleep: bridging the gap between laboratory and epidemiological studies. Sleep Med Rev 2010;14:239–47.
- [37] Piper AT. Sleep duration and life satisfaction. SOEP Papers on Multidisciplinary Panel Data Research 2015. No. 745. http://hdl.handle.net/10419/109135.
- [38] Bixler E. Sleep and society: an epidemiological perspective. Sleep Med 2009;10:S3-6.
- [39] Krueger PM, Friedman EM. Sleep duration in the United States: a crosssectional population-based study. Am J Epidemiol 2009;169:1052–63.
- [40] Luyster FS, Strollo Jr PJ, Zee PC, et al. Boards of directors of the American Academy of sleep M, the sleep research S. Sleep: a health imperative. Sleep 2012;35:727-34.
- [41] Knutson KL, Van Cauter E, Rathouz PJ, et al. Trends in the prevalence of short sleepers in the USA: 1975–2006. Sleep 2010;33:37–45.
- [42] Ohayon MM, Carskadon MA, Guilleminault C, et al. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. Sleep 2004;27: 1255–74.

- [43] Ursin R, Bjorvatn B, Holsten F. Sleep duration, subjective sleep need, and sleep habits of 40-to 45- year-olds in the Hordaland Health Study. Sleep 2005;28: 1260.
- [44] Kronholm E, Puusniekka R, Jokela J, et al. Trends in self-reported sleep problems, tiredness and related school performance among Finnish adolescents from 1984 to 2011. J Sleep Res 2015;24:3–10.
- [45] Uhlig BL, Sand T, Ødegård SS, et al. Prevalence and associated factors of DSM-V insomnia in Norway: the Nord-Trøndelag health study (HUNT 3). Sleep Med 2014;15:708–13.
- [46] Danielsson NS, Harvey AG, MacDonald S, et al. Sleep disturbance and depressive symptoms in adolescence: the role of catastrophic worry. J Youth Adolesc 2013;42:1223–33.
- [47] Johnson EO, Roth T, Schultz L, et al. Epidemiology of DSM-IV insomnia in adolescence: lifetime prevalence, chronicity, and an emergent gender difference. Pediatrics 2006;117:e247–56.
- [48] Lindberg E, Janson C, Gislason T, et al. Sleep disturbances in a young adult population: can gender differences be explained by differences in psychological status? Sleep 1997;20:381–7.
- [49] Pallesen S, Sivertsen B, Nordhus IH, et al. A 10-year trend of insomnia prevalence in the adult Norwegian population. Sleep Med 2014;15:173–9.
- [50] Lamers F, Hickie I, Merikangas KR. Prevalence and correlates of prolonged fatigue in a US sample of adolescents. Am J Psychiatry 2013;170:502–10.
- [51] ter Wolbeek M, van Doornen LJ, Kavelaars A, et al. Severe fatigue in adolescents: a common phenomenon? Pediatrics 2006;117:e1078-86.
- [52] ter Wolbeek M, van Doornen LJ, Kavelaars A, et al. Fatigue, depressive symptoms, and anxiety from adolescence up to young adulthood: a longitudinal study. Brain Behav Immun 2011;25:1249–55.
- [53] Mai E, Buysse DJ. Insomnia: prevalence, impact, pathogenesis, differential diagnosis, and evaluation. Sleep Med Clin 2008;3:167–74.
- [54] Ohayon MM, Reynolds CF. Epidemiological and clinical relevance of insomnia diagnosis algorithms according to the DSM-IV and the International Classification of Sleep Disorders (ICSD). Sleep Med 2009;10:952–60.
- [55] Chung K-F, Yeung W-F, Ho FY-Y, et al. Cross-cultural and comparative epidemiology of insomnia: the diagnostic and statistical manual (DSM), international classification of diseases (ICD) and international classification of sleep disorders (ICSD). Sleep Med 2015;16:477–82.
- [56] Morin CM, Jarrin DC. Epidemiology of insomnia. Sleep Med Clin 2013;8: 281–97.
- [57] Kim MJ, Lee JH, Duffy JF. Circadian rhythm sleep disorders. J Clin outcomes Manag JCOM 2013;20:513.
- [58] Drake CL, Roehrs T, Richardson G, et al. Shift work sleep disorder: prevalence and consequences beyond that of symptomatic day workers. Sleep 2004;27: 1453–62.
- [59] Ohayon MM, Priest RG. Night terrors, sleepwalking, and confusional arousals in the general population: their frequency and relationship to other sleep and mental disorders. J Clin Psychiatry 1999;60(1). 478–276.
- [60] Spoormaker VI. Nightmares: assessment, theory, and treatment. Utrecht University; 2005.
- [61] Slater G, Steier J. Excessive daytime sleepiness in sleep disorders. J Thorac Dis 2012;4:608–16.
- [62] Swanson LM, Arnedt J, Rosekind MR, et al. Sleep disorders and work performance: findings from the 2008 National Sleep Foundation Sleep in America poll. J Sleep Res 2011;20:487–94.
- [63] Walsleben JA, Kapur VK, Newman AB, et al. Sleep and reported daytime sleepiness in normal subjects: the Sleep Heart Health Study. Sleep 2004;27: 293–8.
- [64] Allen RP, Picchietti D, Hening WA, et al. Restless legs syndrome: diagnostic criteria, special considerations, and epidemiology: a report from the restless legs syndrome diagnosis and epidemiology workshop at the National Institutes of Health. Sleep Med 2003;4:101–19.
- [65] Ancoli-Israel S, Shochat T. Insomnia in older adults. In: Kryger M, Roth T, Dement W, editors. Principles and practice of sleep medicine. New York: Elsevier; 2011. p. 1544–50.
- [66] Chokroverty S, Bhat S. Movement disorders in sleep. In: Chokroverty S, Billiary M, editors. Sleep medicine: a comprehensive guide to its development, clinical milestones, and advances in treatment. New York: Springer; 2015. p. 237–48.
- [67] Montplaisir J, Allen RP, Walters AS, et al. Restless legs syndrome and periodic limb movements during sleep. In: Kryger M, Roth T, Dement W, editors. Principles and practice of sleep medicine. New York: Elsevier; 2011. p. 1026–37.
- [68] Allen RP, Stillman P, Myers AJ. Physician-diagnosed restless legs syndrome in a large sample of primary medical care patients in western Europe: prevalence and characteristics. Sleep Med 2010;11:31–7.
- [69] Mahowald MW. Other parasomnias. In: Kryger M, Roth T, Dement W, editors. Principles and practice of sleep medicine. New York: Elsevier; 2011. p. 1098–105.
- [70] De Backer W. Central sleep apnoea, pathogenesis and treatment: an overview and perspective. Eur Respir J 1995;8:1372–83.
- [71] Wellman A, White DP. Central sleep apnea and periodic breathing. In: Kryger M, Roth T, Dement W, editors. Principles and practice of sleep medicine. New York: Elsevier; 2011. p. 1140–52.
- [72] Chowdhuri S, Quan SF, Almeida F, et al. An official American Thoracic society research statement: impact of mild obstructive sleep apnea in adults. Am J Respir Crit Care Med 2016;193:e37–54.