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SCIENTIFIC INVESTIGATIONS

Differences between subjective and objective sleep duration according to actual sleep duration and sleep-disordered breathing: the Nagahama Study

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Study Objectives: Since subjective sleep duration (SSD) is considered to be longer than objective sleep duration (OSD), results of SSD minus OSD (SSD−OSD) might always be thought to be positive. Some recent reports showed different results, but exact results have not been obtained. The difference between SSD and OSD may change according to OSD. We investigated this difference and its association with sleep-disordered breathing (SDB) or nonrestorative sleep.

Methods: This cross-sectional study evaluated 6,908 community residents in Nagahama, Japan. SSD was determined by self-administered questionnaire. OSD was measured by wrist actigraphy and sleep diary. SDB was assessed according to the 3% oxygen desaturation index adjusted for OSD.

Results: Worthy of notice was that SSD was shorter than OSD for those with SSD longer than 6.98 hours in all participants, 7.36 hours in males, and 6.80 hours in females. However, SSD was longer than OSD (mean ± SD: 6.49 ± 1.07 vs 6.01 ± 0.96; $P < .001$) overall, as SSD is considered to be longer than OSD. In patients with SDB, the difference between SSD−OSD was greater when OSD was **s**horter. The difference also depended on SDB severity. The degree of positivity between OSD and SSD was a significant factor in nonrestorative sleep (odds ratio: 2.691; $P < .001$).

Conclusions: When OSD was slightly less than 7 (6.98) hours, participants reported or perceived SSD > OSD. When OSD was > 6.98 hours, participants reported or perceived SSD < OSD. Patients with SDB reported longer SSD than OSD according to severity of SDB. Evaluating SSD, OSD, and their differences may be useful for managing sleep disturbances, including nonrestorative sleep.

Keywords: subjective sleep duration, objective sleep duration, actigraphy, sleep-disordered breathing

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BRIEF SUMMARY

Current Knowledge/Study Rationale: Since subjective sleep duration (SSD) is considered to be longer than objective sleep duration (OSD), SSD minus OSD has been thought to always be positive. But the difference between OSD and SSD may change according to OSD or sleep disturbances such as sleep-disordered breathing. The difference might also be correlated with nonrestorative sleep.

Study Impact: In 6,908 community residents, SSD was shorter than OSD when OSD was longer than 6.98 hours in all participants, 7.36 hours in males, and 6.80 hours in females; and the degree of difference in positivity between OSD and SSD was a significant factor for nonrestorative sleep ($P < .001$). In addition, sleep-disordered breathing and its severity were associated with the difference between SSD and OSD.

INTRODUCTION

Both short and excessively long sleep durations have been associated with disturbances in the quality of life and lifestyle-associated diseases, including obesity, diabetes, hypertension, and other metabolic diseases.[1–5] Therefore, it is important to measure true sleep duration for the management of human health. However, previous data on the relationships between sleep duration and several diseases were usually based on self-reported subjective sleep duration (SSD). Recent technical progress in the measurement of actual sleep time—that is, objective sleep duration (OSD)—revealed substantial differences between SSD and OSD. Subsequently, it was indicated that the results of studies based on SSD might not be reproduced when analyses were done with OSD.[6–8] In addition, previous reports showed that when SSD and OSD increased, these values did not increase proportionally.[9,10]

In general, SSD was reported to be longer than OSD,[11–13] but the results of previous studies implied that the difference between OSD and SSD might not be uniform and could be affected by various factors. Previous studies suggested that the difference might change according to the length of the sleep duration itself or the sex of study participants.[6–10] However, detailed analyses of the characteristics of the differences between SSD and OSD have not been performed, and studies with large-scale cohorts are lacking. Therefore, the characteristics of the difference between SSD and OSD or their sex differences have not been established.

In addition to sex differences, comorbidities may alter the differences between SSD and OSD. Among disorders, sleep-disordered breathing (SDB) may be a major factor for the differences. SDB frequently causes excessive sleepiness,[14,15] which may significantly affect SSD. In addition, arousals by SDB would shorten OSD.[16,17] Nevertheless, little is known about the characteristics of the differences between SSD and OSD in those with SDB.

Recently, nonrestorative sleep (NRS) has become an important issue worldwide.[18] NRS is defined as the self-reported experience of not having been sufficiently refreshed or restored by sleep.[19–21] There have been several studies on insomnia and NRS,[22] insomnia and misperceptions regarding sleep duration (OSD > SSD),[23] insomnia and SDB,[24] and SDB and NRS.[25] But no study has determined whether the misperception that SSD is shorter than OSD (OSD > SSD) might be significantly associated with NRS.

In the present study, we set 2 hypotheses: first, the difference between SSD and OSD might change according to OSD or the severity of SDB and, second, misperceptions, especially OSD > SSD, might have a significant association with NRS. We evaluated the difference between SSD and OSD among participants with differences in length of OSD using data from the Nagahama Study, a large-scale cohort of a general population in Japan.[26,27] We further investigated whether the degrees of differences between SSD and OSD might be altered according to the severity of SDB, which has a significant association with insomnia and NRS.[24,25]

METHODS

Study design and study population

This cross-sectional study was conducted using survey data obtained from 2013 to 2016 from the Nagahama Prospective Genome Cohort for Comprehensive Human Bioscience (the Nagahama Study[28]). The Nagahama Study included residents in Nagahama, a rural city with approximately 125,000 inhabitants in Japan. Nagahama residents aged 30 to 74 years and without serious health problems were recruited for the study's initial phase via mass communications in the local community, such as public relations magazines and periodical newspapers, as well as personal solicitations. Recruited for the second phase were 9,850 participants from 34 to 80 years old without apparent physical impairments or dysfunction. Written informed consent was obtained from all participants. The study protocol was approved by the Kyoto University Graduate School and Faculty of Medicine Ethics Committee (G278).

Assessment of SSD and OSD and SDB

The SSD data were obtained from a self-administered questionnaire. Participants were asked to respond to the Japanese version of the Pittsburgh Sleep Quality Index (PSQI).[29–31] The PSQI includes 7 components with a total of 18 questions. Components were on subjective sleep quality, sleep latency, sleep duration (SSD), sleep efficiency, sleep disturbance, use of sleep medications, and daytime dysfunction. The PSQI was sent to the participants 3 weeks before the days on which actigraphy (see below) was used. Most participants returned the questionnaires 1 week before the assessments began. Therefore, we thought that the SSD reported on the PSQI would be obtained close to the days on which OSD was measured by actigraphy.

We used the answer to the question on sleep duration as the source of the SSD data. OSD was measured by a wrist actigraph and a sleep diary. As previously reported,[32] actigraphy was performed using the Actiwatch 2 or the Actiwatch Spectrum Plus wrist actigraph (Philips Respironics, Murrysville, PA) worn on participants' nondominant wrist for 7 consecutive days. Participants also completed a sleep diary over the same period.[33] Data from a minimum of 4 weekdays and at least 1 weekend day were required for the analysis and were averaged.[34] Bed-in time and bed-out time were set by well-trained investigators and manually confirmed based on sleep diaries and the device's light sensor. Total sleep duration (from sleep-onset time to wake-up time) and actual sleep duration (sleep duration after exclusion of wake time after sleep onset from total sleep duration) were determined using the standard factory-default algorithm. In our study, participants were divided into 2 groups: OSD > SSD and OSD ≤ SSD.

We obtained the actigraphy-modified 3% oxygen desaturation index (Acti-ODI3%) from the 3% oxygen desaturation index and actual sleep duration by actigraphy. Oxygen saturation (SpO_2) was measured by pulse oximetry (PULSOX-Me300; Konica Minolta, Inc., Tokyo, Japan). In addition, we compared the Acti-ODI3% and the apnea-hypopnea index (AHI) derived from attended polysomnography. We previously reported[26] that Acti-ODI3% was more comparable to the AHI derived from attended polysomnography in 32 patients ($r = .99$, $P < .001$; AHI = Acti-ODI3% × 1.04 + 1.45 events/h) than simply measured ODI3% without actigraphy modification ($r = .92$, $P < .001$; AHI = usual ODI3% × 1.27 + 2.06 events/h).[26] The severity of SDB was defined by Acti-ODI3% levels. Acti-ODI3% < 5 events/h was considered as normal; mild SDB was defined as Acti-ODI3% of 5 to < 15 events/h; moderate SDB was defined as Acti-ODI3% of 15 to < 30 events/h; and severe SDB was defined as Acti-ODI3% ≥ 30 events/h.

Assessment of sleepiness

The degree of daytime sleepiness was assessed based on the Japanese version of the Epworth Sleepiness Scale (ESS).[35] The ESS consists of 8 items and can measure subjective daytime excessive sleepiness.[36] Scores between 0 and 10 points are

considered normal,[37] while 11 or more points indicate excessive daytime sleepiness.[38]

Assessment of self-reported NRS

Refreshment or restoration by sleep was assessed by a "yes-no" question in a self-administered questionnaire ("Do you get adequate rest during sleep?"). Individuals who answered "No" were considered to be experiencing NRS, as reported previously.[28]

Comorbidity

In this study, we considered hypertension and diabetes as explanatory variables, which have been shown to be strongly associated with SDB. Participants with the following characteristics were considered as having hypertension: systolic blood pressure (SBP) ≥ 140 mm Hg or diastolic blood pressure (DBP) ≥ 90 mm Hg or taking antihypertensive agents. The presence of diabetes was indicated by glycated hemoglobin (HbA1c) ≥ 6.5 or taking oral hypoglycemic agents and/or insulin.

Statistical analysis

Descriptive statistics were used to assess participants' characteristics. The *t* test, Wilcoxon rank-sum test, 1-way analysis of variance (ANOVA), Bartlett's test, and Kruskal-Wallis test were used to compare continuous values and the chi-square test was used to compare categorical values. A Bland-Altman plot was drawn to show the relationship between the mean of SSD and OSD and the difference between those values. In order to examine what factor was significant in the increase in the difference between SSD and OSD, a multiple regression model was created with the difference between SSD and OSD (SSD−OSD) as the response variable. Age, sex, body mass index, drinking habit, smoking habit, presence or absence of hypertension, presence or absence of diabetes, ESS, and SDB severity were used as explanatory variables. Drinking habit was divided into 2 groups: did not drink at all and did drink. Smoking was divided according to not smoking at all and smoking. Furthermore, a multiple logistic regression model was created with the question, "Do you get adequate rest during sleep?" as the response variable (odds ratio for a "no" answer). Age, sex, severity of SDB, and OSD > SSD status were used as explanatory variables.

Statistical analyses were performed with Stata SE version 14.2 (StataCorp, College Station, TX). All tests were 2-sided, with $P < .05$ considered significant.

RESULTS

Participant characteristics

For this study we examined data on 9,850 individuals from the second-phase cohort (2013–2016) of the Nagahama Study. We excluded data on 1,858 participants for the following reasons: missing actual sleep-related data (n = 1,771), missing SSD data from the questionnaire (n = 76), missing blood pressure data (n = 1), missing biochemical data (n = 1), and missing response to a necessary questionnaire item (n = 9). We also excluded data on 1,084 participants due to an insufficient number of days for which OSD data were available (**Figure 1**).

Figure 1—Flowchart of participant selection.

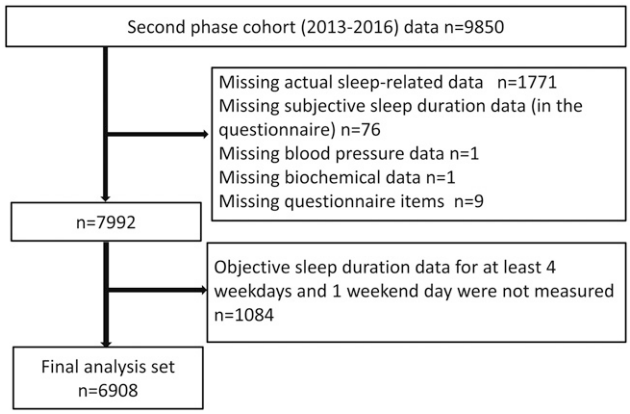


Table 1 shows the final study population (n = 6,908) and the prevalence of OSD > SSD and OSD ≤ SSD according to participants' characteristics. We further divided participants into 5 groups according to the length of OSD: OSD less than 5 hours (OSD < 5 h), OSD from 5 hours or more to less than 6 hours (OSD ≥ 5 h < 6 h), OSD from 6 hours or more to less than 7 hours (OSD ≥ 6 h < 7 h), OSD from 7 hours or more to less than 8 hours (OSD ≥ 7 h < 8 h), and OSD of 8 hours or more (OSD ≥ 8 h) (**Table S1** in the supplemental material).

Comparison of SSD and OSD

We evaluated whether the difference between SSD and OSD changed according to the length of OSD (**Table S1**). We found relatively longer SSD compared with OSD in the groups with OSD < 7 h ($P < .001$). In contrast, SSD tended to be shorter than OSD in the group with OSD ≥ 8 h. When the groups were further stratified according to sex, males with OSD ≥ 7 h < 8 h had longer SSD compared with OSD ($P < .001$) (**Table S1**).

Figure 2 and **Figure S1** in the supplemental material show scatterplots with regression lines based on each participant's OSD and SSD. **Figure 3** shows a Bland-Altman plot between SSD and OSD. Visual inspection of the Bland-Altman plot revealed many data points outside the 95% confidence interval, and the mean was offset, lying above zero. These findings suggested the existence of a systematic error within the 2 methods of measuring sleep duration. Based on this result, we analyzed the discrepancy by delineating the regression lines that estimated SSD from OSD. The analysis of overall participants (**Figure 2:** total; $r = .437$, $P < .001$) and the stratified analysis according to sex (**Figure S1A:** males; $r = .417$, $P < .001$; and **Figure S1B:** females; $r = .454$, $P < .001$) are shown. We performed a single regression analysis treating SSD as an objective variable and OSD as an explanatory variable. The analysis revealed that the coincidence points for the estimated SSD and OSD were 6.98, 7.36, and 6.80 hours for total participants, males, and females, respectively (**Figure 2** and **Figure S1**).

Table 1—Participants with OSD > SSD or OSD ≤ SSD according to participants' characteristics.

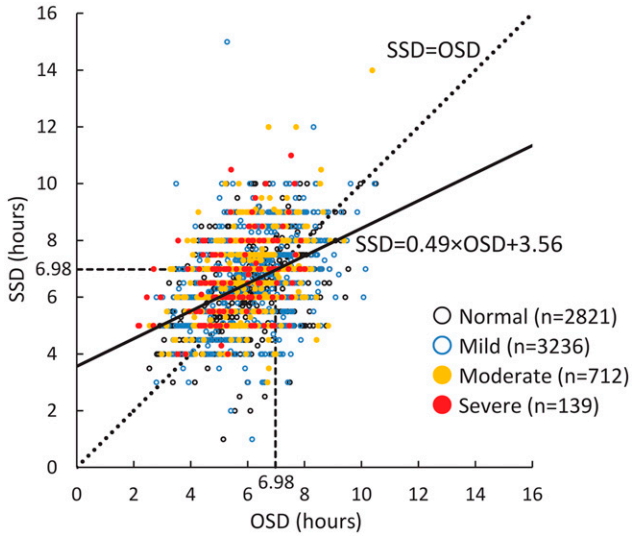
	Total (n = 6,908)	OSD > SSD (n = 2,107)	OSD ≤ SSD (n = 4,801)	*P*
Males	2,241	575 (25.7)	1,666 (74.3)	< .001
Females	4,667	1,532 (32.8)	3,135 (67.2)	
Age (y)	57.9 ± 12.0	58.9 ± 11.7	57.5 ± 12.1	< .001
BMI (kg/m^2)	22.2 ± 3.3	22.0 ± 3.3	22.3 ± 3.3	< .001
Drinking habit				
Yes	3,764	1,128 (30.0)	2,636 (70.0)	.293
No	3,144	979 (31.1)	2,165 (68.9)	
Smoking habit				
Yes	2,127	573 (26.9)	1,554 (73.1)	< .001
No	4,781	1,534 (32.1)	3,247 (67.9)	
Hypertension				
Yes	2,428	774 (31.9)	1,654 (68.1)	.067
No	4,480	1,333 (29.8)	3,147 (70.2)	
Diabetes				
Yes	455	116 (25.5)	339 (74.5)	.016
No	6,453	1,991 (30.9)	4,462 (69.1)	
Number of participants with SDB				
Normal	2,821	944 (33.5)	1,877 (66.5)	< .001
Mild	3,236	957 (29.6)	2,279 (70.4)	
Moderate	712	177 (24.9)	535 (75.1)	
Severe	139	29 (20.9)	110 (79.1)	
SSD (h)	6.49 ± 1.0	5.79 ± 0.90	6.79 ± 0.10	< .001
OSD (h)	6.01 ± 0.96	6.51 ± 0.88	5.79 ± 0.91	< .001
NRS				
Yes	2,616	1,110 (42.4)	1,506 (57.6)	< .001
No	4,292	997 (23.2)	3,295 (76.8)	
ESS score	6.22 ± 4.06	6.28 ± 4.20	6.19 ± 4.00	.396
Acti-ODI3% (events/h)	5.96 [3.67–10.2]	5.42 [3.41–9.14]	6.23 [3.77–10.6]	< .001
SpO_2 (%)	96.7 ± 1.29	96.7 ± 1.27	96.6 ± 1.30	.5305
Min SpO_2 (%)	83.7 ± 5.69	83.8 ± 5.40	83.7 ± 5.81	.3131
CT90 (%)	0.18 [0.05–0.76]	0.16 [0.04–0.62]	0.19 [0.05–0.80]	< .001

Data are expressed as mean ± SD, median [lower–higher interquartile range], or number (% among each group) as appropriate. Severity of SDB was classified by Acti-ODI3% levels as follows: normal, < 5 events/h; mild, 5 to < 15 events/h; moderate, 15 to < 30 events/h; and severe, ≥ 30 events/h. *P* value: chi-square test, *t* test, Wilcoxon rank-sum test. BMI = body mass index, CT90 = cumulative percentage time at SpO_2 below 90%, ESS = Epworth Sleepiness Scale, NRS = nonrestorative sleep, Acti-ODI3% = actigraphy-modified 3% oxygen desaturation index, OSD = objective sleep duration, SD = standard deviation, SDB = sleep-disordered breathing, SpO_2 = percutaneous oxygen saturation, SSD = subjective sleep duration.

Comparison of OSD and SSD according to the level of severity of SDB

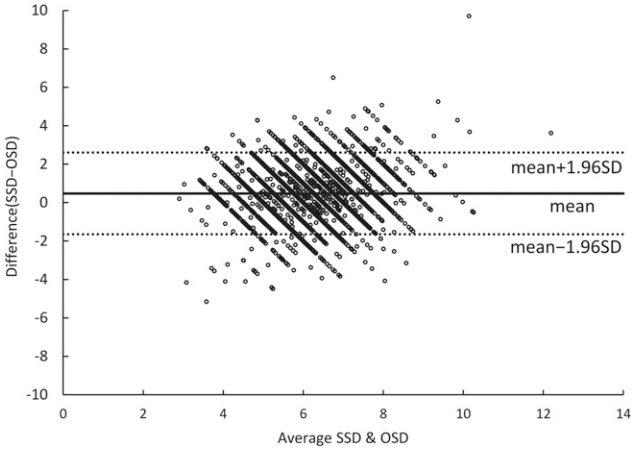
Then we examined whether SDB was associated with the difference between OSD and SSD. As shown in **Table 1**, with increased severity of SDB, the ratio of participants with OSD ≤ SSD increased. Consistently, when the participants were grouped according to the severity of SDB, the difference calculated by subtracting OSD from SSD increased as the severity of SDB escalated (**Table 2**). As we also found that several factors were potentially associated with the difference in OSD and SSD (**Table S2** in the supplemental material), we performed multiple regression testing and confirmed that each level of severity of SDB was a significant factor for the difference between SSD and OSD even after adjusting for participants' characteristics or comorbidities (**Table S3** in the supplemental material). **Figure 4** shows a comparison of regression lines that estimated SSD from OSD according to the severity of SDB. Single regression analysis was performed treating SSD as the objective variable and OSD as the explanatory variable. OSD and SSD were estimated to be equal at values of 6.83, 7.04, 7.35, and 7.38 hours for normal, mild, moderate, and severe SDB, respectively.

Figure 2—Scatterplots for SSD and OSD.



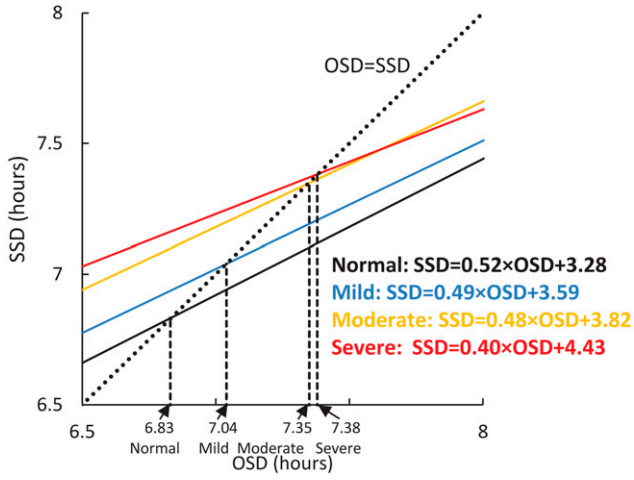
Coincidence points for estimated SSD and OSD were 6.98, 7.36, and 6.80 hours for total, males, and females, respectively (see also **Figure S1**). OSD = objective sleep duration, SSD = subjective sleep duration.

Figure 3—Bland-Altman plot between SSD and OSD.



The difference between SSD and OSD (SSD–OSD) plotted against the mean of SSD and OSD (n = 6,908). A solid horizontal line indicates the mean value of the SSD–OSD difference, and the 2 dotted lines indicate the 95% confidence interval (mean difference ± 1.96 SD). OSD = objective sleep duration, SD = standard deviation, SSD = subjective sleep duration.

Figure 4—Changes in sleep duration when OSD became equal to SSD.



Severity of SDB was classified by Acti-ODI3% levels as follows: normal, < 5 events/h; mild, 5 to < 15 events/h; moderate, 15 to < 30 events/h; and severe, ≥ 30 events/h. The comparison of regression lines for SSD estimated from the OSD among the participants was grouped according to the degree of SDB severity. The equal point (OSD=SSD) was 6.83 hours in participants without SDB. Equal points were prolonged according to increases in severity of SDB. Acti-ODI3% = actigraphy-modified 3% oxygen desaturation index, OSD = objective sleep duration, SDB = sleep-disordered breathing, SSD = subjective sleep duration.

Assessment of the relationship between the SSD−OSD difference and NRS

Table 3 is a summary of the characteristics of NRS groups. To investigate whether the longer OSD compared with SSD was associated with NRS, we compared the rate of participants who answered that they were refreshed or restored by sleep between those with OSD > SSD and OSD ≤ SSD (**Table 1**). Interestingly, participants with OSD > SSD had a higher prevalence of NRS than OSD ≤ SSD (NRS vs non-NRS: OSD > SSD groups 1,110 [52.7%] vs 997 [47.3%]; OSD ≤ SSD groups 1,506 [31.4%] vs 3,295 [68.6%]) (**Table 3** and **Figure 5**). The OSD > SSD status was a significant factor for NRS even after adjusting for participants' characteristics and comorbidities by multiple logistic regression model (**Table S4** in the supplemental material).

Table 2—Comparison of sleep duration parameters between participants grouped according to the severity of SDB.

	Normal	Mild	Moderate	Severe	*P*
SSD	6.39 ± 1.01	6.53 ± 1.07	6.65 ± 1.21	6.66 ± 1.31	< .001
OSD	6.05 ± 0.94	6.02 ± 0.95	5.88 ± 1.04	5.60 ± 1.21	< .001
SSD–OSD	0.34 ± 1.00	0.50 ± 1.08	0.77 ± 1.23	1.07 ± 1.41	< .001

Data are expressed as mean ± SD. Severity of SDB was classified by Acti-ODI3% levels as follows: normal, < 5 events/h; mild, 5 to < 15 events/h; moderate, 15 to < 30 events/h; and severe, ≥ 30 events/h. *P* value: *t* test, 1-way ANOVA. ANOVA = analysis of variance, OSD = objective sleep duration, SD = standard deviation, SDB = sleep-disordered breathing, SSD = subjective sleep duration.

Table 3—Participants with or without NRS according to participants' characteristics.

	NRS (n = 2,616)	Non-NRS (n = 4,292)	*P*
Males	779 (34.8)	1,462 (65.2)	< .001
Females	1,837 (39.4)	2,830 (60.6)	
Age (y)	55.1 ± 11.5	59.6 ± 12.0	< .001
BMI (kg/m^2)	22.2 ± 3.5	22.3 ± 3.1	.358
Drinking habit			
Yes	1,492 (39.6)	2,272 (60.4)	.001
No	1,124 (35.8)	2,020 (64.2)	
Smoking habit			
Yes	770 (36.2)	1,357 (63.8)	.057
No	1,846 (38.6)	2,935 (61.4)	
Hypertension			
Yes	768 (31.6)	1,660 (68.4)	< .001
No	1,848 (41.3)	2,632 (58.8)	
Diabetes			
Yes	135 (29.7)	320 (70.3)	< .001
No	2,481 (38.4)	3,972 (61.6)	
Number of participants with SDB			
Normal	1,167 (41.4)	1,654 (58.6)	< .001
Mild	1,166 (36.0)	2,070 (64.0)	
Moderate	238 (33.4)	474 (66.6)	
Severe	45 (32.4)	94 (67.6)	
SSD (h)	5.99 ± 1.00	6.79 ± 1.00	< .001
OSD (h)	5.81 ± 0.98	6.14 ± 0.93	< .001
SSD < OSD status			
OSD > SSD	1,110 (52.7)	997 (47.3)	< .001
OSD ≤ SSD	1,506 (31.4)	3,295 (68.6)	
ESS score	7.51 ± 4.39	5.43 ± 3.64	< .001
Acti-ODI3% (events/h)	5.53 [3.41–9.57]	6.23 [3.80–10.4]	< .001
SpO_2 (%)	96.8 ± 1.27	96.6 ± 1.30	< .001
Min SpO_2 (%)	84.0 ± 5.70	83.6 ± 5.68	.004
CT90 (%)	0.15 [0.04–0.67]	0.20 [0.05–0.79]	< .001

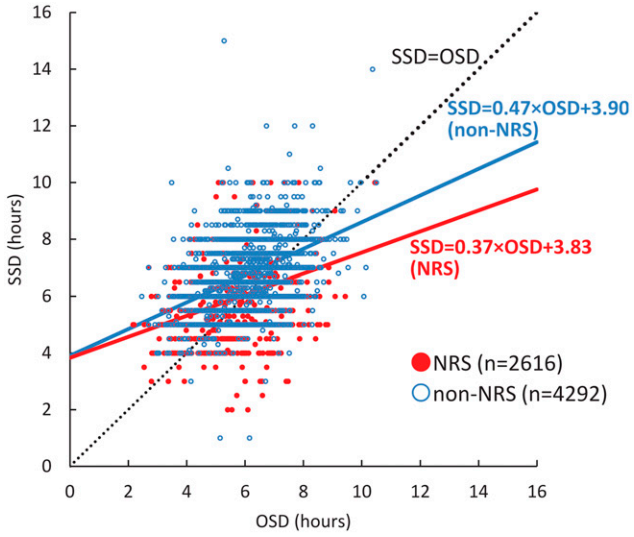
Data are expressed as mean ± SD, median [lower–higher interquartile range], or number (% among each group) as appropriate. Severity of SDB was classified by Acti-ODI3% levels as follows: normal, < 5 events/h; mild, 5 to < 15 events/h; moderate, 15 to < 30 events/h; and severe, ≥ 30 events/h. *P* value: chi-square test, *t* test, Wilcoxon rank-sum test. BMI = body mass index, CT90 = cumulative percentage time at SpO_2 below 90%, ESS = Epworth Sleepiness Scale, NRS = nonrestorative sleep, Acti-ODI3% = actigraphy-modified 3% oxygen desaturation index, OSD = objective sleep duration, SD = standard deviation, SDB = sleep-disordered breathing, SpO_2 = percutaneous oxygen saturation, SSD = subjective sleep duration.

DISCUSSION

In the present study, by using a large-scale community-based cohort, we examined the relationship between SSD and OSD by stratifying participants according to OSD values. As a result, contrary to the general opinion that SSD is longer than OSD, we showed that SSD could exhibit shorter periods than OSD when OSD neared 7 (6.98) hours (females: 6.80 hours; males: 7.36 hours). In addition, we first identified SDB as a factor that had a significant association with the degrees of misperception between SSD and OSD, and that as the severity of SDB worsened, SSD tended to become longer than OSD. Last, OSD longer than SSD (OSD > SSD) was a significant factor in NRS (odds ratio: 2.691; $P < .001$).

Previous studies consistently reported that overall mean SSD tended to be longer than mean OSD,[11–13] as was shown in this report. In this study, mean SSD was 6.49 hours, which was more than 1 hour shorter than in previous reports.[9,10,13] The mean OSD in this study was 6.01 hours, which was nearly equal to that in Black individuals (6.0 hours) or Chinese individuals (6.3 hours) in a recent study.[13] It is well known that the Japanese-reported SSD is among the shortest in the world,[39,40] and this fact was confirmed again in this study.

Figure 5—Scatterplots for SSD and OSD by category of NRS.



Comparison of regression lines for SSD estimated from OSD among the participants in the NRS or non-NRS group. NRS = nonrestorative sleep, OSD = objective sleep duration, SSD = subjective sleep duration.

In the present study, we first identified that SDB further increased the degree of misperception between SSD and OSD. As shown in **Figure 3**, OSD and SSD were estimated to be equal at values of 6.83, 7.04, 7.35, and 7.38 hours for normal, mild, moderate, and severe SDB, respectively. This result suggested that the offset points for SSD and OSD could shift depending on the severity of SDB. Namely, participants were apt to report longer SSD according to the severity of SDB when their OSD became shorter. Usually, SDB-related arousals increase according to the severity of SDB. Increases in arousals might increase the differences between SSD and OSD, especially when the OSD is shorter. Second, participants with SDB might be disturbed about the perception of SSD and would think that their SSD would be longer if their OSD became shorter.[41] From these data, physicians who treat patients with SDB should be aware that patients will often report longer SSD compared with OSD according to the severity of SDB, especially in patients with moderate to severe SDB, at least before treatment. A study to determine whether this discrepancy would change following SDB treatment should be warranted in the future.

In addition, we first found that among participants in this study with OSD ≥ 7 hours, SSD was apt to become shorter compared with OSD. We think that this phenomenon may reflect the disturbance in sleep quality due to long sleep duration,[42–44] or mismatch in perception between OSD and SSD, which could decrease satisfaction obtained from sleep and might shorten perceived SSD. Of interest, this study revealed that a shorter SSD compared with OSD (OSD > SSD) is a significant factor for a higher association with NRS. These results may indicate that not only a short sleep duration but a mismatch when OSD lengthened (OSD > SSD) could induce NRS. There have been several reports about the relationships between insomnia and NRS,[22] insomnia and misperceptions regarding sleep duration (OSD > SSD),[23] insomnia and SDB,[24] and SDB and NRS.[25] In this study, we first reported that OSD > SSD was a factor in NRS in the large cohort. The most recent study[45] showed that misperception of sleep duration is common in sleep laboratory patients, but is most prominent in insomnia.[45,46] We found that the value that OSD equals to SSD (OSD = SSD) increased according to the severity of SDB. On the other hand, insomnia is one of the main symptoms in patients with SDB. Therefore, the prolongation in the value that OSD equals to SSD (OSD = SSD) should be further studied, including the insomnia factor of patients with SDB.

We also found that males and females had different points of time when SSD was equal to OSD. Consistently, the frequency of females was higher in the participants with OSD > SSD and the average levels of SSD–OSD was smaller in females. Therefore, when considering the relationship between SSD and OSD, it may be necessary to consider sex differences.

Our study has several limitations. First, causal inference could not be clarified as this study had a cross-sectional design. Second, the level of SDB was not assessed by polysomnography but by pulse oximetry. However, we calculated the oxygen desaturation index according to objective sleep duration by actigraphy (Acti-ODI3%), which is in close accordance with sleep duration as measured by polysomnography as shown in the Methods section.[26,32] Third, although we did not use polysomnography for measuring objective sleep duration as in a previous study,[13] the results of this study derived from actigraphy and a sleep diary would be meaningful. Fourth, we analyzed a homogeneous ethnic group (only Japanese participants). As shown in previous studies,[39,40] Japanese sleep duration was one of the shortest in the world. Therefore, racial differences may have existed with regard to the results. Fifth, although Nagahama is a small to mid-size city in Japan, it has urban and rural parts. In addition, some of participants work in Kyoto or Osaka, which are metropolitan cities in Japan. In Japan, many persons in rural cities like Nagahama commute to work by train to metropolitan cities. Therefore, we cannot know whether the participants' responses in this study were influenced by regional specificity.

In conclusion, by using a community-based, large-scale cohort in Japan, SSD was shown to be longer than OSD in groups with OSD < 7 hours, as previously reported, while SSD was shorter than OSD in groups with OSD ≥ 7 hours. In addition, we first reported that patients with SDB have longer SSD compared with OSD, especially when OSD was short. In addition, those with a longer OSD than SSD had a higher risk of NRS. These data will help physicians better manage patients' complaints about sleep duration. In future studies, the underlying mechanisms for these findings are expected to be investigated.

ABBREVIATIONS

Acti-ODI3%, actigraphy-modified 3% oxygen desaturation index

AHI, apnea-hypopnea index
ESS, Epworth Sleepiness Scale
NRS, nonrestorative sleep
OSD, objective sleep duration
PSQI, Pittsburgh Sleep Quality Index
SDB, sleep-disordered breathing
SSD, subjective sleep duration

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