Sleep and Normal Subjects

A Longitudinal Study of Laboratory- and Diary-Based Sleep Measures in Healthy “Old Old” and “Young Old” Volunteers

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Summary: The major aim of this study was to examine laboratory- and diary-based measures of sleep in a group of healthy (“successfully aging”) “old old” subjects (≥75 years of age), as contrasted with a group of “young old” subjects (60–74 years of age), who were followed longitudinally for 2 years. We hypothesized that sleep would deteriorate to a greater extent over time among the old old subjects than among the young old. The study group consisted of 50 elders (21 male, 29 female; 23 old old, 27 young old), each studied at baseline and then again at 1- and 2-year follow-up. Analysis of variance was used to determine main effects of age group, gender and time on key sleep measures. Most measures were found to be remarkably stable over time. However, some decay was detected in sleep efficiency among the old old, but not among the young old. In a multiple regression model for the cohort as a whole, age, cognitive status and medical burden at baseline predicted subsequent declines in sleep efficiency over the 2-year period. To our knowledge, this is the first longitudinal data set on sleep in the healthy old old. Key Words: Sleep—Aging—Old old—Healthy elderly—Sleep quality.

Because the elderly are the most rapidly expanding segment of our population, there has been increasing interest in their sleep-related problems, particularly those of the frail and old old group [for review see Roth and Roehrs (1)]. The Epidemiologic Catchment Area (ECA) study reported 6-month prevalence rates of 12–15% for sleep disturbances in persons 65 years of age and older, demonstrating these disturbances to be among the most common health problems for elderly (2). A major public health challenge posed by the increasing proportion of elderly persons in our population is to facilitate their remaining active and well. Otherwise, increased longevity is likely to be associated primarily with prolonged dependency (3). Because changes in sleep and sleep quality in late life have enormous impact on quality of life, level of functioning and ability to remain independent (4–6), preserving the integrity of sleep in late life is an important public health priority, as emphasized by the National Institutes of Health Consensus Development Conference (7).

The majority of published data on the effects of sleep in healthy elderly men and women are derived from cross-sectional studies that have focused primarily on the young old, that is, elders in their 60s and 70s [for review see Reynolds et al. (8)]. We have recently expanded the normative sleep data base to include the old old, or elders 75 years of age and over (8). Published longitudinal reports of sleep in late life have also focused on elders in their 60s and 70s and present different conclusions. For instance, changes in sleep and sleep-disordered breathing measures over time have been reported by some (9–14), but not by all studies (15,16). The current study expands longitudinal sleep data to include the old old.

There is now considerable evidence supporting pathogenic roles for medical, affective and cognitive disorders in the poor sleep of elderly men and women. As recently reviewed by Bliwise (17), medical illness has both direct and indirect effects on sleep in old age (2,10,18). Specific clinical symptoms or disorders have been shown to have negative effects upon sleep quality in late life, including nocturia (19), headache (20), gas-
trointestinal illness (21,22), bronchitis and asthma (22),
and cardiovascular symptoms and type II diabetes (23).
In addition, elevated autonomic activity (24,25) and
a greater susceptibility to external arousal (26,27) may
be important predisposing factors to disturbed sleep
in late life. Conversely, we have also documented that
sleep parameters at baseline recording in high-func-
tioning elders predicted the direction of change in
chronic medical burden scores by follow-up 12 months
later, such that elders with worse sleep at baseline scored
higher on a measure of cumulative medical burden at
1-year follow-up (28).

With respect to affective disorder, persistent sleep
disturbance has been identified in the ECA data base
as a significant risk factor for the subsequent develop-
ment of major depression, as well as a major factor
in decisions to seek services from the primary-care
sector (2). Our own work (29), as well as that of others
(30,31), provides evidence linking affect balance (e.g.
depression) and sleep outcomes in late life.

Finally, cognitive impairment has also been linked
to sleep disturbances among the elderly. We have re-
ported both increases in sleep-disordered breathing as
well as deterioration in sleep continuity and rapid eye
movement (REM) sleep in elderly patients with prob-
able Alzheimer’s dementia (32). These changes also
appear to be correlated with probability of mortality
by 2-year follow-up (33). Similar findings with respect
to the loss of REM sleep in Alzheimer’s dementia have
been reported by Prinz et al. (34) and Vitiello et al.
(35). Ancoli-Israel et al. (36) and Bliwise et al. (37)
have reported high prevalence of sleep apnea with ag-
ing and attendant increases in morbidity and mortality.
Other studies (38-40) have shown that sleep-related
behaviors often precipitate the decision of families to
institutionalize an elderly demented relative.

Longitudinal studies of sleep and sleep quality in
“successful” aging (41) may further elucidate the im-
portance of sleep in maintaining quality of life in old
age, as well as the impact of medical, affective and
cognitive disorders on sleep. To determine whether
and how sleep deteriorates with advancing age in sub-
jects who are aging well with minimal or no functional
impairment, we initiated a study of both laboratory-
and diary-based measures of sleep and sleep quality
among two groups of healthy elderly: old old (those
≥75 years of age) and young old (<75 years) over a
2-year interval. We examined the effects of medical,
affective and cognitive status on sleep over time, as
well as the association of gender with sleep measures.
Our hypotheses were as follows: 1) Sleep will deteri-
orate to a greater degree over time among the old old
subjects than among young old subjects; 2) gender dif-
ferences will remain stable over time, with elderly men
having greater disturbance in measures of sleep con-
tinuity, depth and sleep-disordered breathing than el-
derly women; and 3) medical, affective and cognitive
status will significantly correlate with change in sleep
variables.

METHOD

Subjects

The study group consisted of 50 elders (21 men and
29 women) who volunteered to participate in our sleep
and aging research program. At study entry, subjects’
mean age was 74.6 years (standard deviation 7.7, range
61.1-89.2). Subjects were grouped using the criteria of
Blazer et al. (42) and our previous work (8) as either
old old (≥75 of age, n = 23); or as young old (<75
years, n = 27). Most subjects were high school grad-
uates (92%, averaging 14.9 years of education) and
white (98%). The majority (80%) were retired from
paid employment or were homemakers. All subjects
were living in the community either alone or with
family during the 2-year study interval and were socially
active. During the course of follow-up, two subjects
from the young old group were eliminated from the
study because of noncompliance with protocol pro-
cedures. Five old old subjects resigned from the study
for medical reasons. Most subjects were in the study
group previously described in our cross-sectional
analysis of sleep in old old and young old subjects (8).

At study intake, subjects met stringent physical and
mental health criteria. Routine laboratory tests (in-
cluding complete blood count with differential, levels
of folate, B12, rapid plasma reagin, electrolytes, BUN,
creatinine, total and direct bilirubin, calcium, choles-
terol, amylase, SGOT and SGPT; uric acid; thyroid
profile; urinalysis; electrocardiogram; and chest X-ray),
together with medical and neurological examinations,
were used to screen out individuals with serious pres-
ent or past neurological or other uncontrolled physical
illness, or who were taking medications that could af-
fect sleep or mood. Of the total sample, 19 (38%) were
under a physician’s care for stable medical illnesses
(e.g. heart disease, allergies, hypertension, hypothy-
roid) and were taking related medications (cardiac, n
= 7; allergy, n = 2; antihypertensive, n = 5; thyroid,
n = 2; other, n = 5). These individuals reported that
health conditions had little or no effect on their activ-
ities of daily living. Additionally, at baseline subjects
had no evidence for present or past psychiatric disorder
assessed by the Schedule for Affective Disorders and
Schizophrenia—Lifetime Version (SADS—L) (43).
Hamilton depression ratings (44) were required to be
less than seven (the first 17 items), and the Folstein
Mini-Mental Status (45) rating was required to be 27
or higher at baseline.
Procedure

A uniform procedure for obtaining study data was followed at each of the three study assessments (baseline, 1-year follow-up and 2-year follow-up). Assessment of physical and mental health and subjective sleep quality was completed within 1 month before each set of sleep studies. Subjects kept a diary of daily activities (46) and a sleep-wake log (47) for 2 weeks before coming into the laboratory. Additionally, subjects were asked not to use alcohol for 2 weeks before and during each set of sleep studies. Polysomnography was performed in private bedrooms during each subject's habitual sleep time, as ascertained from the sleep-wake log.

During the day, subjects were free to leave the laboratory and carry on their usual daily activities. However, three subjects were enrolled in a protocol that required them to remain in the laboratory's apartment-like living area during the 3-day period (48). In addition, subjects were contacted via telephone at the 6-month interval between sleep studies to ascertain stability or change in health and sleep habits from the time of baseline sleep studies and to foster subjects' continued interest in study participation.

Laboratory-based sleep measures

At each series of sleep studies, subjects had 3 consecutive nights of polysomnography, including 1 night of monitoring for sleep-disordered breathing and periodic limb movements. Electroencephalogram (EEG) sleep variables of sleep continuity, sleep architecture and REM sleep were derived from data averaged over nights 2 and 3, with night 1 considered an adaptation night. Sleep was scored in 60-second epochs following the standard criteria outlined by Rechtschaffen and Kales (49) by polysomnographic technologists who maintain an 85% interrater reliability using procedures previously published (50). Airflow was measured via nasal and oral thermistors, and respiratory effort was measured with bellows. Oxygen saturations were measured via finger probe with the Ohmeda 3700 Biox Oximeter. Hypopnea was scored if airflow during sleep decreased to less than ½ of baseline (amplitude of airflow during the 2-minute period prior to the event) for at least 10 seconds, and apnea was scored if airflow was absent for at least 10 seconds (51). The apnea-hypopnea index (AHI) was computed as the ratio of events to total sleep time in hours. Oxygen saturations were analyzed using a microcomputer PROFOX program, which defines the onset of a desaturation event when the saturation decreases four or more percentage points within a 2-minute interval. Periodic limb movements were scored according to the criteria of Coleman (52).

Diary-based sleep measures

Measures of habitual risetime and habitual bedtime were determined for each subject during the 14-day period preceding each laboratory-based assessment (46). Subjective sleep quality for the month preceding each laboratory assessment was assessed by the Pittsburgh Sleep Quality Index (PSQI) (53,54).

Medical, affective and cognitive measures

The Cumulative Illness Rating Scale (CIRS) (55), as modified by Miller et al. (56) for specific use with geriatric subjects, was utilized to quantify chronic medical burden (higher scores indicating greater medical burden). Depressive symptoms were evaluated at each assessment with the 17-item Hamilton Rating Scale for Depression (single rater). Cognitive status was assessed with the Folstein Mini-Mental Status examination.

Analyses

The study group was stratified by gender and age to test for gender differences as well as age-dependent differences between the young old and the old old in both laboratory-based and diary-based sleep measures. Means and standard deviations for key sleep variables across subjects were calculated at each of the three time points: baseline, 1-year follow-up and 2-year follow-up. Prior to analyses, distributions of all variables were examined. Natural log transformations were performed to normalize the distributions of sleep latency and sleep efficiency; square-root transformations were used to stabilize variance for REM latency, REM percent, delta percent and REM density.

To test hypotheses 1 and 2, analyses focused on comparisons between young old and old old subjects, and between men and women in these age groups, across the three assessment points. Thus, for each key sleep variable, separate three-way analyses of variance (ANOVAs) were performed. Each ANOVA included two between-subjects factors for age (young old vs. old old) and gender (male vs. female), and one within-subjects factor for time of assessment.

To address hypothesis 3 (i.e. relationships between change in sleep variables and baseline measures of medical, affective and cognitive status), we selected sleep variables that were either not stable over time, as assessed with the ANOVAs, or were of critical importance conceptually or clinically. We computed cross-sectional correlations between baseline predictors.
(CIRS, Hamilton and Folstein scores) and baseline sleep variables, as well as longitudinal correlations of Time-1 predictors with Time-2 and Time-3 sleep variables. Cross-sectional correlations between CIRS and sleep efficiency were computed at Time 2 and Time 3 for both young old and old old subjects. The homogeneity of correlations procedure was used to test the strength of cross-sectional and longitudinal correlations. In addition, forced multiple regression procedures using baseline measures of age, chronic medical burden (CIRS), AHI, myoclonus index, affective status (Hamilton) and cognitive status (Folstein) as independent variables were used to model changes in key sleep measures such as sleep efficiency and sleep quality (PSQI), in order to understand the relative contributions of each predictor.

RESULTS

Hypothesis 1: Sleep will deteriorate to a greater degree over time among old old subjects than among young old subjects

Laboratory-based sleep measures (Table 1)

In general, sleep continuity indices were remarkably stable over the 2-year observation period. However, sleep efficiency declined among subjects ≥75 years of age but remained stable in the young old group (time interaction F(2,90) = 4.91, p < 0.01). Effects of age group per se were detected in sleep efficiency (decreased in subjects ≥75 years old, p < 0.01). Only one measure of sleep architecture, REM sleep percent, differed between age groups (less among the old old, p < 0.03). REM sleep measures were otherwise stable over time in both groups.

The extent of sleep-disordered breathing and nocturnal myoclonus for both age groups was minimal. Median AHI increased over time (p < 0.002), but no specific age effects or age × time interactions were detected.

Diary-based sleep measures (Table 1)

Diary-based measures of habitual bedtime and rise-time were stable over 2-year follow-up, with 7.5-8 hours total in bed. No effects of age or gender were detected. However, there appeared to be consistently greater variability in the habitual bedtime of the old old subjects, as reflected in larger standard deviations. Sleep quality (PSQI) diminished over 2 years (effect of time; p < 0.01) and showed greater impairment among the old old than young old subjects (group effect; p < 0.04). The absence of a significant time group inter-action effect indicates a stable difference in sleep quality over time between the two age groups.

Hypothesis 2: Gender differences will remain stable over time, with elderly men having greater disturbance in measures of sleep continuity, depth and sleep-disordered breathing than elderly women

Gender effects were evident in percent of slow-wave sleep (decreased in men; p < 0.01), REM latency (decreased in men; p < 0.01) and AHI (greater in men; p < 0.001). These effects remained stable over time. As noted above, gender was not related to diary-based measures, and no gender × time interaction effects were detected for major sleep variables, such as sleep efficiency and slow-wave sleep percent. Gender × group effects were evident in total recording period (p < 0.003), sleep latency (p < 0.04), time spent asleep (p < 0.04), REM latency (p < 0.001) and habitual rise-time (p < 0.04).

Hypothesis 3: Medical, affective and cognitive status will significantly correlate with change in sleep variables

As shown in Table 2, medical burden (CIRS) increased over 2 years (effect of time significant; p < 0.0001), and this increase was larger among subjects ≥75 years of age than among those <75 (age group × time interaction; p < 0.0001). Men and women did not differ in medical burden over time or in measures of mental health.

As shown in Table 3, when health variables were examined as potential predictors of degree of sleep efficiency, the CIRS measures of medical burden at baseline were related to both concurrent sleep efficiency (r = 0.70) and subsequent sleep efficiency at Time 2 (r = 0.56, p < 0.008) in the old old subjects. Thus higher medical burden predicted lower sleep efficiency in this age group (the correlation with sleep efficiency is positive because we used natural-log transformation of 100 - sleep efficiency in the analysis). Additionally, cross-sectional correlations between CIRS and sleep efficiency were significant at Time 2 among the old old subjects (r = 0.39, p < 0.05). Using the homogeneity of correlations procedures, there was no difference detected in strength between the significant longitudinal relationship and the cross-sectional relationship (χ² = 0.59, ns). In contrast, CIRS was only weakly associated with sleep efficiency in younger subjects. The same health variables (CIRS, Hamilton and Folstein scores) were examined as potential predictors of subjective sleep quality (PSQI). The correlation between CIRS score at baseline and PSQI score at baseline was not
 TABLE 1. Laboratory- and diary-based sleep, sleep-disordered breathing and myoclonus measures: Mean (SD) across age groups and time of assessment

<table>
<thead>
<tr>
<th>Subjects &lt;75 years (n = 27)</th>
<th>Subjects ≥75 years (n = 23)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
</tr>
<tr>
<td>Polysomnographic measures</td>
<td></td>
</tr>
<tr>
<td>Total recording period (minutes)</td>
<td>456.3 (48.8)</td>
</tr>
<tr>
<td>Sleep latency (minutes)</td>
<td>17.1 (12.3)</td>
</tr>
<tr>
<td>Awake (minutes)</td>
<td>60.6 (37.2)</td>
</tr>
<tr>
<td>Time spent asleep (minutes)</td>
<td>378.6 (40.5)</td>
</tr>
<tr>
<td>Sleep efficiency (%)</td>
<td>83.2 (7.0)</td>
</tr>
<tr>
<td>Stage 1 (%)</td>
<td>6.1 (4.1)</td>
</tr>
<tr>
<td>Stage 2 (%)</td>
<td>65.6 (8.2)</td>
</tr>
<tr>
<td>Delta (%)</td>
<td>5.4 (6.8)</td>
</tr>
<tr>
<td>REM (%)</td>
<td>22.9 (4.7)</td>
</tr>
<tr>
<td>REM latency (minutes)</td>
<td>51.6 (15.1)</td>
</tr>
<tr>
<td>REM density (units/minute)</td>
<td>1.5 (0.6)</td>
</tr>
<tr>
<td>Apnea measures</td>
<td></td>
</tr>
<tr>
<td>Apnea-hypopnea index (events/hour)</td>
<td>3.5 (5.4)</td>
</tr>
<tr>
<td>Median</td>
<td>2.1</td>
</tr>
<tr>
<td>Apnea index (events/hour)</td>
<td>1.3 (2.2)</td>
</tr>
<tr>
<td>Median</td>
<td>0.1</td>
</tr>
<tr>
<td>Oxygen desaturation</td>
<td></td>
</tr>
<tr>
<td>Lowest desaturation (%)</td>
<td>86.3 (4.5)</td>
</tr>
<tr>
<td>Myoclonus measures</td>
<td></td>
</tr>
<tr>
<td>Myoclonus index (events/hour)</td>
<td>8.6 (13.4)</td>
</tr>
<tr>
<td>Diary-based measures</td>
<td></td>
</tr>
<tr>
<td>Habitual sleep times</td>
<td></td>
</tr>
<tr>
<td>Habitual bedtime (time of day)</td>
<td>2308 (0.38)</td>
</tr>
<tr>
<td>Habitual risetime (time of day)</td>
<td>0700 (1:11)</td>
</tr>
<tr>
<td>PSQI</td>
<td>3.4 (1.8)</td>
</tr>
</tbody>
</table>

Significance levels of repeated measures ANOVA:
* F = 9.57, df = 1.45, p < 0.003, Age group·Sex.
* F = 4.71, df = 1.45, p < 0.04, Age group·Sex (transformed using natural log).
* F = 4.34, df = 1.45, p < 0.04, Sex; F = 4.53, df = 2.90, p < 0.01, Time·Age group; F = 3.41, df = 2.90, p < 0.04, Time·Sex (transformed using square root).
* F = 10.16, df = 1.45, p < 0.003, Age group; F = 8.49, df = 1.45, p < 0.006, Sex; F = 4.58, df = 1.45, p < 0.04, Age group·Sex.
* F = 7.31, df = 1.45, p < 0.01, Age group; F = 6.88, df = 1.45, p < 0.01, Sex; F = 3.06, df = 2.90, p < 0.05, Time; F = 4.91, df = 2.90, p < 0.01, Time·Age group (transformed using natural log).
* F = 5.73, df = 1.45, p < 0.02, Age group; F = 14.04, df = 1.45, p < 0.0005, Sex; F = 4.62, df = 2.90, p < 0.01, Time (transformed using square root).
* F = 4.11, df = 2.90, p < 0.02, Time·Sex.
* F = 35.62, df = 1.45, p < 0.0001, Sex (transformed using square root).
* F = 4.78, df = 1.45, p < 0.03, Age group.
* F = 3.92, df = 1.45, p < 0.05, Age group; F = 14.18, df = 1.45, p < 0.0005, Sex; F = 12.09, df = 1.45, p < 0.001, Age group·Sex (transformed using square root).
* χ² using the CATMOD procedure with a cutoff of five events per hour.
* χ² = 12.87, df = 1, p < 0.002, Time.
* F = 3.98, df = 2.48, p < 0.03, Time.
* F = 6.51, df = 1.39, p < 0.01, Age group·Sex.
* Pittsburgh Sleep Quality Index: F = 4.74, df = 1.41, p < 0.04, Age group; F = 4.57, df = 2.82, p < 0.01, Time.

significant (r = 0.29, ns). CIRS score at baseline was related to subsequent sleep quality at assessment Time 2 (r = 0.51, p < 0.05) and Time 3 (r = 0.55, p < 0.05) in the old old subjects. Correlations were not significant among the young old subjects. The zero-order correlations of the Hamilton depression score and Folstein Mini-Mental score with both sleep efficiency and sleep quality were small and insignificant in both cross-sectional and longitudinal analyses.

Because these predictors were intercorrelated, however, a forced multiple regression model was applied that included baseline measures of age, medical burden, affective state, cognitive status, apnea-hypopnea index and myoclonus index as predictors of sleep change. We considered change in sleep efficiency and PSQI as separate outcome variables. The six predictors did not explain change of subjective sleep quality (PSQI) over the 2-year follow-up [F(6,36) = 1.86, p < 0.11]. However, the forced multiple regression to predict total change in sleep efficiency (over 2 years) in the sample showed, in contrast, that there were three significant predictors: increased age (standardized beta = −0.38,
DISCUSSION

The current study breaks new ground in two important areas. First, it is based on a longitudinal data set including both laboratory- and diary-based measures of sleep among successfully aging elders studied at three assessments 12 months apart. Second, the study examined and compared rates of change in sleep in important areas. First, it is based on a longitudinal data set including both laboratory- and diary-based measures of sleep among successfully aging elders studied at three assessments 12 months apart. Second, the study examined and compared rates of change in sleep in important areas.

Our primary finding is that the majority of sleep measures showed remarkable stability across the 2-year study period in both the young old and old old. We have previously suggested that stability of EEG sleep measures in late life (particularly REM sleep measures) provides a strong correlate of successful and stable adaptation in the elderly (57). The current results are consistent with this concept and extend it to elders of even relatively advanced age. However, an alternative explanation for the relative stability of the sleep measures from our elderly volunteers is that their 2-year membership in the research program itself had an effect. It should be stated that the program was not intended to be a treatment or education program, and it included no explicit components in that regard. However, we did develop a good working relationship with our volunteer subjects and provided regular health screenings. Moreover, as a sleep program, we probably caused our subjects to become more aware of sleep-related issues.

Even so, differences did emerge over time with respect to sleep efficiency and sleep quality, with declines in the old old, but not in the young old. These observations then led us to ask what other variables might best predict these observed changes in sleep, whether such predictors would be equally strong across the full range of elderly years, or whether some predictors would be stronger harbingers of poor sleep in the old old than in the young old. First, concerning sleep efficiency, our correlational analyses (Table 3) showed that medical burden at baseline was significantly related to poorer concurrent and subsequent level of sleep efficiency only in the old old, not in the young old. Our multiple regression examining change in sleep efficiency showed that increasing age, decreasing cognitive status and increasing medical burden were significant predictors of decline in sleep efficiency. This issue may be important from a clinical intervention standpoint. For example, our data suggest that chronic medical burden provides a strong correlate of successful and stable adaptation in the elderly. Our finding that decay in sleep efficiency in healthy nondemented elders was related to decreasing cognitive status (Folstein) appears to converge with earlier published observations in patients with probable demen-

### TABLE 2. Medical, affective and cognitive measures: Mean (SD) across age groups and time of assessment

<table>
<thead>
<tr>
<th>Variable</th>
<th>Subjects &lt;75 years (n = 27)</th>
<th>Subjects ≥75 years (n = 23)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline  Time 2  Time 3</td>
<td>Baseline  Time 2  Time 3</td>
</tr>
<tr>
<td>Cumulative Illness Rating Scale*</td>
<td>4.0 (2.4)  4.5 (2.8)  5.0 (3.0)</td>
<td>6.3 (3.3)  7.4 (4.0)  8.7 (4.0)</td>
</tr>
<tr>
<td>Hamilton Depression Rating*</td>
<td>0.7 (1.0)  0.7 (0.8)  1.0 (1.0)</td>
<td>1.5 (1.8)  2.1 (2.0)  2.5 (2.0)</td>
</tr>
<tr>
<td>Folstein Mini-Mental Scale</td>
<td>29.7 (0.7)  29.6 (0.6)  29.6 (0.7)</td>
<td>29.2 (1.0)  29.2 (0.8)  29.2 (1.1)</td>
</tr>
</tbody>
</table>

Significance levels of repeated measures ANOVA:

* $F = 12.07, df = 1.45, p < 0.001$, Age group; $F = 3.99, df = 1.45, p < 0.05$, Age group; Sex; $F = 10.73, df = 2.90, p < 0.0001$, Time; $F = 14.14, df = 1.44, p < 0.0005$, Age group.

** $F = 8.34, df = 1.41, p < 0.006$, Age group.

p < 0.02; variance explained = 10%, reduced Folstein scores (beta = 0.34, p < 0.04; variance explained = 8%) and increased medical burden as measured by the CIRS (beta = 0.32, p < 0.058; variance explained = 7%), the $R^2$ of 0.33 for the model for sleep efficiency was significant [F(6,38) = 3.11, p < 0.02].

### TABLE 3. Longitudinal correlations of sleep efficiency with baseline medical, affective and cognitive measures

<table>
<thead>
<tr>
<th></th>
<th>Sleep efficiency [ln(100 - sleep efficiency + 1)]</th>
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<tbody>
<tr>
<td></td>
<td>Baseline  Time 2  Time 3</td>
</tr>
<tr>
<td><strong>Young old</strong></td>
<td></td>
</tr>
<tr>
<td>Cumulative Illness Rating Scale</td>
<td>-0.14  -0.12  -0.14</td>
</tr>
<tr>
<td>Hamilton Depression Rating</td>
<td>0.15  -0.08  0.14</td>
</tr>
<tr>
<td>Folstein Mini-Mental Scale</td>
<td>0.15  -0.09  -0.23</td>
</tr>
<tr>
<td><strong>Old old</strong></td>
<td></td>
</tr>
<tr>
<td>Cumulative Illness Rating Scale</td>
<td>0.70**  0.56*  0.22</td>
</tr>
<tr>
<td>Hamilton Depression Rating</td>
<td>-0.14  0.14  0.05</td>
</tr>
<tr>
<td>Folstein Mini-Mental Scale</td>
<td>-0.08  -0.08  -0.12</td>
</tr>
</tbody>
</table>

Note: due to the transformation of sleep efficiency [ln(100 - sleep efficiency + 1)], a positive correlation indicates that as medical burden increases, sleep efficiency decreases.

* $p < 0.01$.

** $p < 0.001$. 

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mentia of the Alzheimer type that showed that sleep efficiency deteriorates with progression of dementia (32,34-36). Thus, even in nondemented elders, relatively small changes in cognitive status may be associated with physiological deterioration in the continuity of sleep, further affecting elders' life quality.

Subjective sleep quality (PSQI) also showed some slippage over time and slightly greater impairment in subjects older than 75. In this context, old old subjects, concurrent with diminishing sleep efficiency, showed greater variability on some aspects of habitual sleep scheduling. The subjective decline in sleep quality was associated with increase in medical burden. This finding further confirms the negative impact of medical illness on sleep quality in late life (19-23). Clearly this area needs further exploration.

The general impression conveyed by these data is one of accelerated decay in the subjective quality and physiological efficiency of sleep in very late life, paralleling increases in medical burden and cognitive impairment, as well as greater variability in sleep schedule. Despite these modest changes in overall sleep continuity, however, the generation of slow-wave sleep and REM sleep remained remarkably stable over time. This continuity parallels the continuing generally good adjustment of initially healthy elders. Similarly, stable gender effects on slow-wave sleep, REM latency and sleep-disordered breathing were confirmed.

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