Sleep and Sleep Disorders in Older Persons

A Multicomponent Nonpharmacological Intervention Improves Activity Rhythms Among Nursing Home Residents With Disrupted Sleep/Wake Patterns

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Background. Sleep and circadian rhythms are disrupted among many nursing home (NH) residents. We examined the impact of a multicomponent nonpharmacological intervention on 24-hour rest/activity rhythms among long-stay NH residents.

Methods. The study was a randomized controlled trial in which, following a 3-day baseline, participants received 5 days of either usual care (control condition) or the active intervention. The intervention combined increased exposure to outdoor bright light, efforts to keep residents out of bed during the day, structured physical activity, institution of a bedtime routine, and efforts to reduce nighttime noise and light in residents’ rooms. For 100 residents with baseline and follow-up wrist actigraphy data (mean age = 87 years; 76% women), rest/activity rhythms were modeled to determine the rhythm acrophase (peak time), nadir (trough time), midline estimating statistic of rhythm (MESOR) (midpoint), amplitude (height of peak), slope, and the rest period/active period ratio ($\alpha$).

Results. The intervention led to an increase in the duration of the “active” portion of the rhythm, which was primarily accounted for by a shift in the rest/activity rhythm rise to an earlier time. Findings persisted when analyses were adjusted for age, cognitive functioning, medical comorbidities, and behavioral disturbances.

Conclusions. These findings suggest that the intervention may effectively improve the robustness of rest/activity rhythms in NH residents. Further research is needed to examine the impact of similar interventions on other measures of circadian rhythms (e.g., body temperature, melatonin) among NH residents.

An extensive literature shows that community-dwelling older adults with health problems or psychiatric illness have more disturbed sleep than do healthy older people (1), and individuals living in nursing homes (NHs) suffer from even more disturbed sleep than do older individuals living in the community. Prior research has shown that many older people in the NH setting have extremely fragmented nighttime sleep and are excessively sleepy during daytime hours (2,6).

In addition to medical and psychiatric illnesses, several other factors impact sleep among NH residents, including disruption of underlying circadian rhythms. Circadian rhythms are biological rhythms of approximately 24 hours. Among NH residents, abnormalities have been found in body temperature, melatonin, and rest/activity rhythms (2,7,8). Disruption of circadian rhythms results both from underlying physiological abnormalities in the circadian system and from external environmental factors that adversely impact the circadian system (2,9–11).

In younger adults, circadian rhythms are adequately entrained (i.e., synchronized with the environment) by zeitgebers (time cues) encountered during everyday activities, particularly by exposure to light. Older community-dwelling adults are exposed to less bright light than are younger adults, and NH residents are exposed to even less (11,12). Older adults also may respond differently to light than may younger adults, requiring more light to remain entrained (13,14).

In addition to attenuated daytime bright light exposure, NH residents experience a reduction in other potentially entraining environmental factors, including a lack of daytime physical activity and frequent exposure to light at night (6,11,15). Lights left on all night may suppress melatonin levels and increase nighttime wakefulness (16,17).

Studies have reported that interventions involving increasing daytime light exposure among NH residents impact both nighttime sleep quality and rest/activity rhythms (18–21). These studies, however, generally involved the use of indoor light (e.g., bright light boxes), sometimes requiring 1:1 attention from research staff to insure exposure. This requirement limits the generalizability of these interventions to “real-world” NH settings. Studies have examined the impact of physical activity on sleep, and show some promise as well (22–24).
This randomized controlled trial tested the effectiveness of a multicomponent nonpharmacological intervention to improve sleep/wake patterns and rest/activity rhythms among NH residents. This intervention included daily exposure to at least 30 minutes of outdoor sunlight, a standardized program to increase physical activity, institution of a regular bedtime routine, and efforts to reduce nighttime noise and light in residents’ rooms. We previously reported that this comprehensive intervention led to modest improvements in nighttime sleep quality (reduced duration of nighttime awakenings), substantially increased daytime wakefulness, and increased participation in both physical and social activities during the daytime hours (25). We have also reported that, at baseline, more daytime sleeping, less nighttime sleep, and the use of antipsychotic medications were all associated with weaker rest/activity rhythms, and more bright light exposure was related to later activity rhythm peaks (26). The results reported here include analysis of rest/activity rhythms among a subgroup of participants in this larger study (i.e., persons with complete actigraphic records at both baseline and follow-up assessments).

Here, for the first time, we report the effects of this multicomponent intervention on 24-hour rest/activity rhythms in intervention participants versus controls. We hypothesized that participants in the intervention group would show improved rest/activity rhythm quality, including increased rhythm amplitude, more rapid rest-to-activity transitions, and a longer period of activity relative to rest. As the timing of light exposure varied, we did not expect that the intervention would lead to an overall mean shift in the rest/activity rhythm acrophase time.

**METHODS**

**Participants and Settings**

Participants were 118 long-stay residents of four Los Angeles area NHs. Facilities were studied consecutively from 1999 through 2001. At each facility, all residents aged 65 years or older who were not bed bound or in contact isolation were considered for participation. Over a 1–2 month period, a two-step screening process was undertaken to identify residents with both daytime sleepiness and nighttime sleep disruption. This process has been described in detail previously (25,26).

Briefly, in Step 1, excessive daytime sleeping (defined as being asleep during ≥15% of observations) was assessed using behavioral observations (3) performed by research staff every 15 minutes between 9:00 AM and 5:00 PM for 2 days for all residents (n = 473 residents screened). Of the 321 residents (68%) who met this criterion, 196 (61%) residents (or their responsible party) agreed to further screening. In Step 2, participants underwent 2 nights of wrist actigraphy (Minimotionlogger; Ambulatory Monitoring Inc., Ardsley, NY) to estimate the percentage of time asleep between 10:00 PM and 6:00 AM (determined by a validated, automatic scoring algorithm in the ActionW software, AMI) (27). Those who were asleep ≤80% of the time (n = 145) were then enrolled in the study.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Intervention (N = 54)</th>
<th>Control (N = 46)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>87.8 (7.9)</td>
<td>86.3 (10.4)</td>
<td>.40</td>
</tr>
<tr>
<td>% Female</td>
<td>76%</td>
<td>80%</td>
<td>.56</td>
</tr>
<tr>
<td>% Non-Hispanic white</td>
<td>91%</td>
<td>94%</td>
<td>.22</td>
</tr>
<tr>
<td>No. of years in nursing home</td>
<td>2.7 (2.4)</td>
<td>3.5 (3.5)</td>
<td>.19</td>
</tr>
<tr>
<td>Body mass index</td>
<td>24.4 (4.5)</td>
<td>24.6 (5.0)</td>
<td>.88</td>
</tr>
<tr>
<td>Nocturnal oxygen desaturation index*</td>
<td>8.4 (11.0)</td>
<td>5.7 (7.4)</td>
<td>.18</td>
</tr>
<tr>
<td>Routine medications, No./day</td>
<td>7.4 (5.0)</td>
<td>7.9 (4.9)</td>
<td>.65</td>
</tr>
<tr>
<td>PRN medications, No./day</td>
<td>0.4 (0.7)</td>
<td>0.2 (0.6)</td>
<td>.95</td>
</tr>
<tr>
<td>Mini-Mental State Examination score</td>
<td>11.4 (9.4)</td>
<td>10.4 (10.3)</td>
<td>.64</td>
</tr>
<tr>
<td>Cornell Scale for Depression in Dementia score</td>
<td>3.5 (3.5)</td>
<td>4.1 (3.5)</td>
<td>.36</td>
</tr>
<tr>
<td>Cumulative Illness Rating Scale-Geriatrics score</td>
<td>25.0 (5.4)</td>
<td>24.3 (4.9)</td>
<td>.49</td>
</tr>
<tr>
<td>Multidimensional Observation Scale for the Elderly score</td>
<td>103.1 (22.9)</td>
<td>98.6 (26.2)</td>
<td>.39</td>
</tr>
<tr>
<td>Nursing Home Behavior Problem Scale score</td>
<td>20.2 (14.6)</td>
<td>18.6 (14.5)</td>
<td>.66</td>
</tr>
</tbody>
</table>

Notes: *Desaturation events per hour of nighttime recording to estimate presence of sleep-disordered breathing. Details of these data have been published previously (28).

SD = standard deviation; PRN = “as needed” medication.

Twenty-seven residents died or withdrew prior to randomization, leaving 118 residents who were randomized to receive the multicomponent intervention (n = 62) or usual care (control condition, n = 56). Ten participants withdrew or died after randomization, leaving 108 participants who completed the intervention phase (n = 58 intervention, n = 50 control). Of those residents who completed the study, valid actigraphy records were available at baseline and follow-up testing for 54 intervention participants and 46 control participants. The length of actigraphy files used in analyses ranged from 1.7 to 3 days, and 99% (99/100) had at least 2.5 days of actigraphy data available for analysis at both time points. This rate of data loss (7%) is similar to that reported by other NH researchers, and did not differ between treatment groups (19). Characteristics of the 100 residents included in the analyses presented here are summarized in Table 1.

Informed consent was obtained for all participants. For residents who were unable to provide self-consent, informed consent was obtained from their responsible party, with assent of the resident. Research methods were approved by the University of California, Los Angeles Office for the Protection of Research Subjects, and a Single Project Assurance was completed at each NH.

**Procedures**

Following initial screening (outlined above), residents completed a comprehensive battery of clinical measures...
including the Mini-Mental State Examination (MMSE) (29) [a 20-item measure of general cognitive functioning, assessing 5 cognitive domains; score range = 0–30; scores < 24 suggest cognitive impairment]; the Cornell Scale for Depression in Dementia (CSDD) (30) [a 19-item measure of depression in dementia completed by a trained research nurse or study physician; score range = 0–38; higher scores indicate more depressive symptoms]; and the Cumulative Illness Rating Scale-Geriatrics (CIRS-G) (31) [a 14-item measure of illness comorbidity based on structured medical record review and physical examination; score range = 0–56; higher scores indicate greater illness severity].

Nursing staff were interviewed using the Multidimensional Observation Scale for the Elderly (MOSES) (32) [a 40-item scale of multidimensional functioning; score range = 40–160; higher scores indicate more functional impairment] and the Nursing Home Behavior Problem Scale (NHBPS) (33) [a 29-item scale to assess frequency of behavioral disruption; score range = 0–116; higher scores indicate more frequent behavioral disturbances]. Participant height, weight, and medications received during the study were obtained from medical records.

Participants underwent three consecutive days (72 hours) of wrist actigraphy, behavioral observations, and bedside noise and light monitoring at baseline, which was repeated at follow-up under usual care conditions (control group) or with the intervention in place (intervention group; during the final 3 days of the 5-day intervention). To avoid contamination of the control condition, follow-up testing was performed in control participants prior to the initiation of the intervention at each study site. This scheduling of the control condition prior to the intervention condition resulted in a shorter average delay between baseline and follow-up in the control versus intervention groups (16 vs 32 days; \( t = -7.29, p = .0006 \)). For all assessment data, “nighttime” was defined as 10:00 PM to 6:00 AM and “daytime” was defined as 8:00 AM to 8:00 PM.

After baseline assessments were completed at each NH site, participants were randomly allocated to intervention or control groups, without blocking or stratification. Fifty-three percent of enrolled participants were assigned to the intervention group, as we anticipated greater dropout/loss of participants in the control versus intervention groups (16 vs 32 days; \( t = -6.90, p < .0005 \)) and the model fit in this study was similar (mean F = 228 vs 211, \( p > .10 \)), for simplicity and clarity we report the results of rest/activity rhythm modeling using the maximum per-minute activity level. Results were consistent when the average per-minute activity was examined.

Rest/activity rhythms were modeled using a 5-parameter extension of the traditional cosinor analysis (18,19,34–36). This 5-parameter model is an antigonoidal transformation of the cosine curve, and it allows for estimation of parameters describing the shape of the rest/activity rhythm (assuming a period of 24 hours). This model provides distinct advantages over the use of a simple cosine for modeling the rhythm parameters. This method is described in great detail by Marler and colleagues (34). Briefly, for the sigmooidally transformed cosine curves, let \( c(t) = \cos((t - \phi)2\pi/24) \). Then the Hill-transformed cosine curve is \( h(c(t)) = [c(t) + 1]^p/(m^p + [c(t) + 1]^p) \); the antigonoidal-transformed cosine curve is \( \ell(c(t)) = \exp(\beta[\cos(t - \alpha)]/(1 + \exp(\beta[\cos(t - \alpha)])) \); and the antarcotangent-transformed cosine curve is \( \psi(c(t)) = \tan^{-1}(\beta(c(t) - \alpha))/\pi + 1/2 \). The sigmooidally transformed cosine models of the data are given by: \( r(t) = \min + \text{amp} \cdot h(c(t)) \); \( r(t) = \min + \text{amp} \cdot \ell(c(t)) \); \( r(t) = \min + \text{amp} \cdot \psi(c(t)) \), where \( \phi \) is the time of day of the peak (acrophase), \( \min \) is the minimum value of the function, and \( \text{amp} \) (amplitude) is the difference between the minimum and maximum value of the function (because the transformed cosine ranges from 0 to 1). The parameter \( \beta \) determines whether the function \( r(t) \) rises and falls more steeply than does the cosine curve (i.e., large values of \( \beta \) produce curves that are nearly square waves). When \( r(t) \) has approximately a square-wave appearance, \( \phi \) will represent the center of activity.
the “flat” region at the top of the curve. The parameter $\alpha$ determines whether the peaks of the curve are wider than the troughs (i.e., when $\alpha$ is small, the troughs are narrow and the peaks are wide; when $\alpha$ is large, the troughs are wide and the peaks are narrow). A measure analogous to the mid-line estimating statistic of rhythm (MESOR) of the cosine model can also be obtained (i.e., $\text{mes} = \text{min} + \text{amp}/2$).

The parameters in the model analyzed here include the rhythm minimum or nadir ($\text{min; trough of the rhythm}$), amplitude ($\text{amp}; maximum peak of the rhythm}$), acrophase ($\phi$; time of rhythm maximum), $\beta$ (steepness of rise and fall of curve), and $\alpha$ (relative width of peak and trough, or rest/active period ratio). The MESOR (i.e., midpoint between peak and trough) was also calculated, and the point at which the curve crossed the MESOR value was used to describe the $\alpha$ parameter (the relative width of the peak and trough at the MESOR). An overall F-statistic for the model was calculated. This F-statistic is interpreted here as a measure of the model’s “fit” to the data and is viewed as a measure of overall rhythmicity, with robust rhythms yielding higher F values. Additional measures were calculated based on this model to further describe rhythm characteristics, including the time at which the curve reached the MESOR on the ascending and descending portions of the curve. This method, including the mathematical formulae for the parameters, has been described in detail elsewhere (18,19,35,36). All participants had statistically significant rest/activity rhythms at baseline and in follow-up (F values $> 10.8$, $df = 4$, $p < .01$), suggesting that parameter estimates were valid.

Data with nonnormal distributions underwent normal score transformation. In an intent-to-treat analysis, 2 (baseline vs follow-up) × 2 (intervention vs control), split-plot analysis of variance (ANOVA) was used to test for differential changes in rest/activity rhythm variables (described above). For significant interaction effects, Bonferroni-corrected follow-up $t$ tests were performed within each group ($\alpha = .025$). Models were tested with and without age, MMSE score, CIRS-G score, and NHBPS score as covariates. These variables were chosen as covariates based on their theoretical impact on rest/activity rhythms, significant correlations with the rest/activity rhythm parameters, and data meeting the assumptions required for analysis of covariance (ANCOVA) (37). Two-sided testing was performed, with significance at $p < .05$.

**Results**

Based on light sensors in the wrist actigraphs, daytime mean light levels and minutes of exposure to bright light ($\text{lux} > 1000$) were higher during treatment than during baseline in the intervention group, with no change in the control group ($F = 53.8$, $p < .001$). Outdoor light exposure occurred in the morning (before 12:00 PM) on 62% of intervention days, after 12:00 PM on 25% of days, and both before and after 12:00 PM on 13% of days. The timing of the light exposure administration was unrelated to changes in rest/activity rhythms. Participants in the intervention group, on average, spent 19% less time during the day (9:00 AM to 5:00 PM) in bed, whereas there was minimal change in the control participants (2% reduction in time in bed; $F = 30.8$, $p < .001$). Participants in the intervention group received, on average, 46 minutes (standard deviation = 11 minutes) of physical activity divided over 3.1 (standard deviation = 0.4) sessions per day.

There was a significant group (intervention vs control) × study phase (baseline vs follow-up) interaction for the rest/activity rhythm $\alpha$ parameter (relative width of peak and trough; see Figure 1; $F_{1,98} = 5.40$, $p = .022$). Follow-up tests showed that the intervention group had a nonsignificant widening of the rest/activity rhythm peak ($-0.20$ to $-0.04$; $p = .555$), whereas the control group showed a nonsignificant delay (i.e., later shift) of the trough ($-0.19$ to $-0.15$; lower values indicate a wider peak). This interaction was statistically significant when age, MMSE, CIRS-G, and NHBPS scores were included in the model ($F_{1,61} = 4.26$, $p = .043$). There was also a significant group × study phase interaction for the time at which the curve crossed the MESOR (Figure 1B; $F_{1,98} = 5.39$, $p = .022$). Follow-up tests showed that there was a nonsignificant advance (i.e., earlier shift) of the rest/activity rhythm increase in the intervention group (6:49 AM to 6:03 AM; $p = .19$) and a nonsignificant delay (i.e., later shift) in the control group (6:12–6:49 AM; $p = .055$). This interaction was statistically significant when age, MMSE, CIRS-G, and NHBPS scores were included as covariates ($F_{1,61} = 4.26$, $p = .046$). There were no significant group × study phase interactions for other rhythm parameters tested, including the rhythm minimum, amplitude, acrophase, $\beta$ (steepness) parameter, or MESOR.

**Discussion**

This study reports the impact of a comprehensive, nonpharmacological intervention on 24-hour rest/activity...
rhythms among older people living in NHs. Results suggest that the intervention impacted primarily the “active” period of the rest/activity cycles. This is shown by the increase in the amount of time the rest/activity rhythm was above the MESOR. Interestingly, it appears that the control group deteriorated slightly (showing a shorter “active” period), whereas the intervention group improved slightly. Such findings are common in the NH setting, where most individuals show deteriorating health over time and interventions partially ameliorate the deterioration rather than leading to robust improvements over baseline.

The increase in the “active” period of the rest/activity rhythm resulted from a slight advance (i.e., shift to an earlier time) in the time at which the activity levels rose in the morning in the intervention group compared to a slight delay in the control group. Based on the phase-response curve to light exposure, this is the expected direction of change for morning light exposure (38–40). Despite the age-related advance (shift to an earlier time) in circadian rhythms reported in healthy older people (41), Harper and colleagues have found that patients with Alzheimer’s disease show a delay (shift to a later time) in circadian rhythms (42,43). Ancoli-Israel and colleagues (18,19) completed two randomized controlled trials of bright light therapy (using artificial light boxes) in the NH and found that rest/activity rhythms were delayed by morning light therapy in this setting. Importantly, one of these studies included only NH residents with AD (19). Their finding of a delay in activity rhythms with morning light is consistent with these patients having phase-delayed rhythms. Their second study included all residents of the NHs, whether or not these individuals had sleep disturbance at baseline (18). Contrary to these findings, our results showed that outdoor light exposure (primarily but not exclusively in the morning hours) led to a shift in circadian rhythms in the opposite direction, that is, the intervention group showed an advance in circadian rhythms relative to the controls. Our findings are consistent with the traditionally described phase-response curve to light, which shows that morning light should advance circadian rhythms (38–40). In addition, our study included only individuals with sleep disruption at baseline, which may have led to the selection of residents with more phase-advanced endogenous circadian rhythms than those studied by Ancoli-Israel and colleagues.

This study has several limitations. First, only wrist actigraphy is available for estimation of underlying circadian rhythms, and the rhythms described by actigraphy are only a proxy for underlying circadian rhythmicity. The impact of the intervention on other circadian rhythms is not known. Additionally, we present here only one of several viable methods for modeling and analysis of rest/activity rhythms (44–46). Other methods may yield slightly different results. Also, in an effort to increase the generalizability of the intervention to community NH settings, some scientific rigor was sacrificed in terms of light exposure timing; however, detailed data were collected on timing of light exposure activities, and we did not find that there were differential impacts of light based on the timing of the exposure.

Results from this study suggest that a multicomponent approach may improve rest/activity rhythms among NH residents. Future research should examine the underlying changes in circadian rhythms of hormones and body temperature associated with improving environmental conditions, increasing exposure to light, and increasing activity levels during the daytime hours.

Acknowledgments

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References


