Psychoneuroendocrinology (2012) 37, 1029—1038

Available online at www.sciencedirect.com

SciVerse ScienceDirect

journal homepage: www.elsevier.com/locate/psyneuen

Sleep duration buffers diurnal cortisol increases in older adulthood

Rebecca Rueggeberg [a,*], Carsten Wrosch [a,*], Gregory E. Miller [b]

[a] Concordia University, Montreal, Canada
[b] University of British Columbia, Vancouver, Canada

Received 5 April 2011; received in revised form 11 November 2011; accepted 28 November 2011

KEYWORDS
Cortisol secretion;
Sleep duration;
Older adulthood

Summary This study examined the long-term associations between reports of sleep duration and diurnal cortisol secretion in older adulthood. It was hypothesized that longer sleep would protect older adults against increases in diurnal cortisol secretion over time. We tested this hypothesis using three waves of data from a 4-year longitudinal study involving 157 older adults. Results from growth curve and cross-lagged panel analyses demonstrated that levels and increases in sleep duration buffered long-term elevations of diurnal cortisol secretion. Reversed analyses indicated that diurnal cortisol secretion did not predict changes in sleep duration over time. These results were independent from sociodemographic characteristics (i.e., age, sex, partnership status, and education) and health-related variables (i.e., chronic illness, medication usage, body mass index, and smoking). They suggest that long sleep exerts restorative functions and protects older adults from exhibiting increases in diurnal cortisol secretion over time.

Cortisol is a hormone that is secreted by the hypothalamic—pituitary—adrenocortical (HPA) axis. Across the day, cortisol secretion follows a circadian rhythm, reaching the highest levels shortly after awakening and progressively declining until bedtime (Van Cauter and Turek, 1994). Cortisol has widespread regulatory influences in the body which include the mobilization of glucose, the regulation of fluid volume, the modulation of immune function, and various central nervous system effects on behaviors related to feeding, sleeping, and learning (Sapolsky et al., 2000). However, physical or psychological stressors can disrupt the HPA axis homeostasis (Heim et al., 2000; Miller et al., 2007; Schaeffer and Baum, 1984; Weiner, 1992). When patterns of cortisol release become dysregulated, either through increased or decreased output, there are likely to be adverse physiological implications, which may alter vulnerability to various mental and physical illnesses (Bjorntorp and Rosmond, 1999; Lupien et al., 2009; Raison and Miller, 2003).

Age-related elevations in cortisol have been found both in natural settings and in response to laboratory-induced challenge (Almeida et al., 2009; McEwen and Stellar, 1993; Kern et al., 1996; Otte et al., 2005; Sapolsky, 1992; Van Cauter et al., 1996, 2000; Wrosch et al., 2008; McEwen and Stellar, 1993). These processes may occur because common

* Corresponding authors at: Concordia University, Department of Psychology, 7141 Sherbrooke Street West, Montreal, QC, H4B 1R6 Canada.
E-mail addresses: r.rueggeberg@gmail.com, r.rueggeberg@gmail.com (R. Rueggeberg), carsten.wrosch@concordia.ca (C. Wrosch).

0306-4530/$ — see front matter © 2011 Elsevier Ltd. All rights reserved.
doi:10.1016/j.psyneuen.2011.11.012

age-related challenges (e.g., health threats or loss of resources, Baltes et al., 1979; Heckhausen et al., 2010) can trigger emotional distress and increase the release of cortisol into the circulation (Wrosch et al., 2004). In addition, age effects on cortisol secretion may reflect an impairment of the HPA axis feedback control and could underlie a constellation of dysregulation in other bodily systems (Boscaro et al., 1998; McEwen and Sapolsky, 1995; Seeman and Robbins, 1994; Wilkinson et al., 1997). However, despite the importance of understanding the age-related trajectories in HPA axis functioning, a consistent pattern of associations between cortisol level and age has not been established. Several studies observed considerable variability in the age-related changes of cortisol secretion (Lupien et al., 1998, 2005; Seeman et al., 1997), and other research did not find an association between diurnal cortisol output and age (e.g., Greenspan et al., 1993).

To provide an explanation for this inconsistent pattern of findings, we suggest that diurnal cortisol may not increase among all older adults, and that some of them are protected from exhibiting cortisol increases. In addition, such individual differences could be reliably associated with behavioral factors that are likely to modulate cortisol release. To this end, we suggest that sleep duration could be one such factor that has the potential to influence trajectories of older adults' diurnal cortisol secretion (Balbo et al., 2010). Preliminary support for this argument stems from cross-sectional research demonstrating that shorter, as compared to longer, sleep duration was associated with higher cortisol levels across day in a cohort of adults (Kumari et al., 2009). Moreover, chronic short sleepers have been found to exhibit higher nocturnal cortisol levels than chronic long sleepers (Späth-Schwalbe et al., 1992). Further, extant laboratory studies have indicated that experimental sleep restriction can predict increases in cortisol levels both during the night of acute sleep loss (Weibel et al., 1995; Weitzman et al., 1983) and, if wakefulness was prolonged, during the following day (Leproult et al., 1997; Spiegel et al., 1999; Van Cauter et al., 2000). Finally, there is evidence from earlier waves from the same longitudinal study as presented here, suggesting that adaptive sleep patterns can attenuate the adverse effects of elevated cortisol levels on older adults' physical health. These health benefits have been explained by the possibility that the restorative function of sleep can down-regulate high levels of cortisol over time (Wrosch et al., 2008).

We note that the link between cortisol and sleep is likely bi-directional. Indeed, there is evidence that cortisol can be associated with changes in sleep patterns, although such effects have not been found consistently across studies (for a review, see Steiger, 2003). These mixed findings suggest that cortisol may play an indirect role in causing sleeping problems, in which it needs to interact with other biological processes to have effects (for a review, see Buckley and Schatzberg, 2005; Shaver et al., 2002).

Together, the reported research suggests that long sleep could buffer age-related increases in diurnal cortisol secretion, whereas shorter sleep may lead to increases in older adults' cortisol output. Further, such restorative functions of long sleep may be especially important in older adulthood when age-related challenges contribute to psychological and biological disturbances (Wrosch et al., 2007, 2008), and older adults could benefit from adaptive behavioral patterns that support homeostasis. Finally, because cortisol could also influence changes in sleep patterns, conclusions about the direction of effects cannot be drawn from cross-sectional evidence, and the existing short-term longitudinal studies leave unexamined whether sleep duration contributes to sustained increases of cortisol secretion. Thus, longitudinal research is needed that tracks changes in individuals' sleep duration and cortisol output over a prolonged period of time to examine the long-term associations between older adults' sleep duration and diurnal cortisol secretion.

To address this gap in the literature, the present research examined the long-term associations between reports of sleep duration and diurnal cortisol secretion in older adulthood. We expected that cortisol levels would generally increase over time, and that this effect could be moderated by older adults' sleep duration. More specifically, we hypothesized that shorter sleep (and declines in sleep duration) would amplify age-related increases in subsequent cortisol secretion, whereas longer sleep (and increases in sleep duration) would protect against them. In addition, we considered the possibility that both levels of and changes in cortisol could also contribute to changes in sleep duration over time.

Methods

Participants

The study is based on a 4-year longitudinal sample of community-dwelling older adults who took part in the *Montreal Aging and Health Study* (MAHS; Wrosch et al., 2007). Participants were recruited through newspaper advertisement. The only inclusion criterion was that participants had to be older than 60 years because we were interested in examining a normative sample of older adults.

In 2004, we conducted the first wave of the MAHS by assessing a heterogeneous sample of 215 older adults from the Montreal area. The 2-year follow-up included 184 participants and 164 subjects participated in the 4-year follow-up. Reasons for non-participation were being deceased ($n = 13$), having problems that prevented participation ($n = 17$), refusing further participation ($n = 8$), and being unable to locate participants ($n = 13$). Seven additional participants were excluded from the analyses because they did not participate in the 2-year follow-up. Thus, the final sample included 157 participants. Study attrition over 4 years was not significantly associated with baseline measures of the study variables, except for participants' age. Older participants were more likely to discontinue their study participation, $t(213) = -2.30$, $p < .05$.

Materials

The analyses incorporated repeated measures of participants' diurnal cortisol rhythms and sleep duration. In addition, we included a number of sociodemographic (i.e., age, sex, partnership status, and education) and health-related covariates (i.e., chronic illness, medication usage, body mass index, and smoking; see Table 1 for means, standard deviations, and frequencies of main study variables).

Table 1 Means, standard deviations, and frequencies of main study variables (n = 157).

Constructs	Mean (SD) or percentage[a]
Cortisol (AUC) (log nmol/L h)	
4 years	13.45 (2.66)
2 years	13.22 (2.47)
Baseline	12.71 (2.59)
Sleep duration (T1) (h)	
4 years	6.94 (1.49)
2 years	6.83 (1.34)
Baseline	6.69 (1.37)
Age (years)	71.72 (5.55)
Male (%)	48.40
Number of chronic health problems (baseline)	2.26 (1.63)
No chronic health problems (%)	10.80
One chronic health problem (%)	24.80
Two or three chronic health problems (%)	43.90
Four or more chronic health problems (%)	20.50
Cortisol-related medication (%)	80.30
BMI (baseline)	25.69 (3.73)
Underweight (BMI $\leq$ 18.4) (%)	2.50
Normal weight (18.5 $\leq$ BMI $\leq$ 24.9) (%)	38.90
Overweight (25 $\leq$ BMI $\leq$ 29.9) (%)	49.70
Obese (BMI $\geq$ 30) (%)	8.90
Current smoker (%)	10.20
Education (baseline)	2.09 (1.07)

[a] Mean and standard deviation (SD) are presented for continuous variables. AUC = area under the curve; BMI = body mass index. Education was indexed as 0 = no education, 1 = high school, 2 = trade, 3 = masters or doctorate.

Diurnal cortisol rhythms were assessed across waves on three non-consecutive typical days. We asked the participants to collect saliva samples as they engaged in their normal daily activities. On each of the 3 days, the participants collected five saliva samples (by using salivettes) at specific times of the day: awakening, 30 min after awakening, 2 p.m., 4 p.m., and before bedtime. Participants were asked not to eat or brush their teeth immediately prior to saliva collection to prevent contamination with food or blood. The actual time of day was recorded by the participants for all of the collected saliva samples. They were provided with a timer that they had to set at 30 min at the time they collected their first saliva sample after awakening. Compliance with the 30 min measure was generally good, as indicated by small deviations from 30 min after wakening at T1 (M = 3.51 min), T2 (M = 4.65 min), and T3 (M = 2.45 min). To ensure compliance concerning the collection of the afternoon and evening samples, participants were called at 2 p.m. and 4 p.m. They were further instructed to collect the last sample of the day by themselves at the time they went to bed. The saliva samples were stored in participants' home refrigerators until they were returned to the lab 2–3 days after collection was completed (for stability of cortisol concentrations in these conditions, see Clements and Parker, 1998). After the saliva containers were returned to the lab, they were frozen until the completion of the study. Cortisol analysis was performed at the University of Trier, in duplicate, using a time-resolved fluorescence immunoassay with a cortisol–biotin conjugate as a tracer (Dressendörfer et al., 1992). The intra-assay coefficient of variation was less than 5%; the inter-assay variability from cortisol analyses performed at the University of Trier has been found to be routinely below 10%.

All raw cortisol values were log-transformed to obtain normally distributed cortisol data. Total diurnal cortisol secretion was indexed by calculating the area under the curve (AUC) for each collection day using trapezoidal estimation (based on hours after awakening). The AUC was chosen as the outcome for this report because, of the various daytime cortisol metrics, it is the best proxy for overall tissue exposure to the hormone, and thus most likely to relate to sleep patterns and other distal health outcomes (Rodenbeck et al., 2002). Given that some saliva samples may have been contaminated with blood or food, we excluded samples across the three waves of data collection because they deviated more than 3 SDs from the mean cortisol secretion for the time of day (1.2%). In cases in which a single saliva sample was missing, we replaced the missing value with the sample mean before calculating AUC. Within each wave, AUC estimates were averaged across collection days to obtain a stable indicator of individual differences in diurnal cortisol secretion.

Sleep duration was measured using items from the Brief Pittsburgh Sleep Quality Index (Buysse et al., 1989). In all three assessments, we asked the participants to report for the majority of recent days and nights during the past month (a) the time they usually laid down to go to sleep, (b) the time they usually got out of bed in the morning, (c) how long it took them to fall asleep after they had laid down to go to sleep, (d) how many minutes of sleep they had lost because they woke up in the middle of the night, and (e) how many minutes of sleep they had lost because they woke up earlier than their usual time to get up. Global indicators of sleep duration for each of the three assessments were calculated by subtracting the minutes individuals spent in bed during the night without sleeping from the total minutes individuals spent in bed during the night.

Covariates. To minimize spurious associations, we controlled for variables that have been shown in previous research to be associated with cortisol secretion or sleep duration (Gangwisch et al., 2006; Patel et al., 2006; Wrosch et al., 2009). The covariates included baseline levels of participants' age, sex, partnership status, education, chronic illness, cortisol-related medication usage, body mass index, and smoking. Partnership status was coded as 1 (being married or cohabitating) and 2 (being separated, divorced, or widowed). Education was measured by asking participants to report their highest educational degree completed (0 = none, 1 = high school, 2 = trade, 3 = undergraduate degree, 4 = graduate degree). Usage of medications that could be associated with cortisol secretion was assessed by counting the number of medications that either contained glucocorticoids and/or can influence the HPA-axis activity (e.g., antidepressants, β-blockers, or anti-inflammatory drugs). Body mass index was calculated in kg/m^2. Smoking was indexed as whether or not participants used cigarettes daily. Levels of chronic illness were measured by asking participants to report whether they were affected by 17 different health problems (e.g., coronary heart disease, cancer, high blood

pressure, or arthritis). A count variable was computed to obtain an indicator of how many different chronic illnesses each participant experienced.

Data analyses

To test the study's hypotheses, we performed two sets of analyses. The first set applied cross-lagged panel analyses and examined with four regression models whether previous levels of sleep duration would predict subsequent 2-year changes in diurnal cortisol secretion (and whether levels of cortisol would predict 2-year changes in sleep duration). The analyses were controlled for the previously described covariates.

The second set applied growth-curve analysis (utilizing HLM 6.0, Raudenbush et al., 2004) to examine whether cortisol levels would increase over 4-years of study, and whether 2-year increases in sleep duration would ameliorate this effect. We used growth curve analysis because these models allowed us to examine within-person changes across time and to identify between-person predictors of individual differences in within-person changes (Bryk and Raudenbush, 1987). More specifically, we estimated in the Level-1 model the within-person variability in participants' cortisol secretion over 4 years (using data from T1, T2, and T3) as a function of years since study entry and a residual term. In the subsequent Level-2 model, we examined whether 2-year changes in sleep duration (and baseline levels of sleep duration and the covariates) would predict individuals differences in longitudinal trajectories obtained in the Level-1 model. A measure of 2-year changes in sleep duration was obtained in a regression analysis, which predicted sleep duration assessed at T2 from the baseline indicator of sleep duration, and saved the standardized residuals. In order to shed further light on the temporal associations between sleep duration and cortisol secretion, we finally reversed the analyses to examine whether 2-year increases in cortisol levels (using residualized change scores) would predict 4-year changes in sleep duration.

Results

Sample description and preliminary analyses

Table 1 provides a description of the sample. Participants were on average about 72 years old and approximately half of the sample was female. They reported an average of 2—3 chronic health problems, and their mean BMI was at the intersection of normal weight to overweight. Eighty percent of the participants reported that they used one or more medications that either contained glucocorticoids and/or can influence the HPA-axis activity (e.g., antidepressants, β-blockers, or anti-inflammatory drugs). Thirty-six percent obtained an undergraduate degree or a higher education, and the minority of the sample smoked. The socio-demographic characteristics, health status, and medication use obtained in the sample were within the normative range for older adults residing at home (Aging NACO, 2006; Rotermann, 2006).

Participants slept at night on average between 402 min (baseline) and 417 min (4-year follow-up) and their sleep ranged across waves from 212 to 592 min. Measures of sleep duration were significantly correlated with each other across waves (rs = .55—.56, $ps < .01$). In addition, Fig. 1 illustrates the raw cortisol values. The sample as a whole exhibited typical patterns of cortisol secretion in all three study waves, demonstrating high cortisol levels at awakening, peaking 30 min after awakening, and continuously decreasing over the later part of the day until bedtime. The calculated day-to-day AUC values were considerably correlated within each wave of data-collection (rs = .46—.67, $ps < .01$), and averaged AUC estimates were significantly correlated across waves (rs = .30—.45, $ps < .01$).

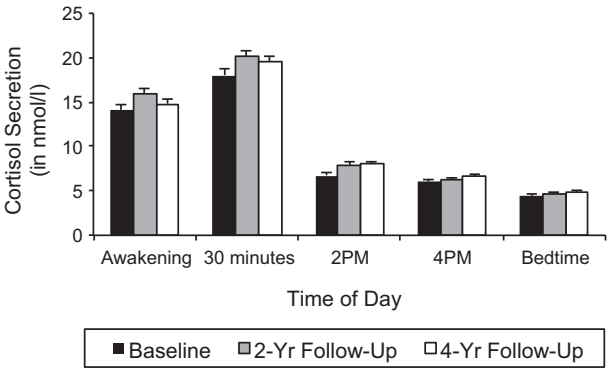

Fig. 1 Means and standard errors of salivary cortisol secretion across three assessment days at baseline, 2-year, and 4-year follow-up.

2-year associations between sleep duration and cortisol secretion

We conducted cross-lagged panel analyses with four separate regression models to examine whether baseline and 2-year levels of sleep duration would predict subsequent 2-year changes in cortisol secretion or vice versa. The results of the analyses are illustrated in Fig. 2 and showed that 2-year, and 4-year levels of the outcome variables were significantly associated with previous levels of the outcomes. While these findings indicate considerable stability in cortisol and sleep duration over time, they also demonstrate that higher baseline levels of sleep duration were associated with smaller 2-year increases in cortisol secretion, $F(1, 147) = 5.95$, $R^2 = .03$, $p < .05$, and higher 2-year levels of sleep duration predicted smaller increases in cortisol secretion over the subsequent 2-year period, $F(1, 147) = 6.98$, $R^2 = .04$, $p < .01$. However, neither baseline levels nor 2-year levels of cortisol secretion were significantly associated with subsequent changes in sleep duration, Fs(1, 147) < 3.44, R^2s < .01, $ps > .05$.[1,2]

[1] We note that the pattern of results obtained in our analyses remained identical if individual differences in compliance with the 30-min measure of cortisol were taken into account. In addition, subsequent analyses showed that neither neuroticism, perceived stress, depressive symptomatology (CES-D), a dichotomous distinction between taking versus not taking any cortisol-related medicine, or any of the 17 assessed chronic health problems (if analyzed separately) explained the reported effects.

[2] 9.6% of our participants took sleeping pills, and follow-up analyses demonstrated that the usage of this medication did not affect the reported associations between sleep and cortisol.

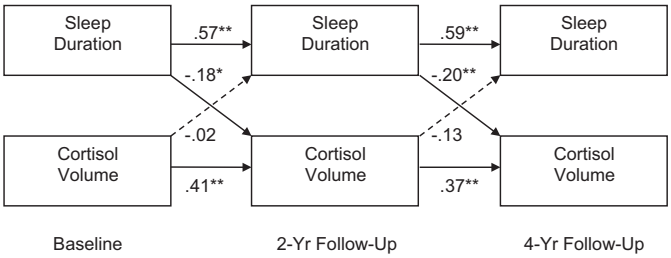

Fig. 2 Cross-lagged panel analyses examining the longitudinal associations between baseline, 2-year, and 4-year levels of sleep duration and cortisol secretion. Values represent standardized regression coefficients.

Predicting 4-year trajectories of cortisol secretion

We conducted growth-curve models to examine whether cortisol would increase over 4 years of study and whether these increases could be moderated by 2-year changes in sleep duration. The results of these analyses are summarized in Table 2 and show that baseline levels of cortisol secretion (i.e., Level-1 intercept) were significantly different from zero. Moreover, the Level-1 slope was significantly positive, which implies that levels of cortisol secretion exhibited a linear increase over time. Finally, there was significant variability around the averaged Level-1 intercept and slope, χ^2s(156) > 223, *ps* $< .01$, indicating the presence of reliable individual differences in baseline levels of, and changes in, cortisol secretion.

In the Level 2-model, we attempted to explain this variability and predicted individual differences in Level-1 intercept and slope by the centered baseline scores of sleep duration, 2-year changes in sleep duration, covariates, and a random residual term. The results from the Level-2 model revealed that baseline levels of sleep duration were negatively associated with baseline levels of cortisol secretion (see effects on intercept in Table 2), indicating that participants who reported shorter sleep duration at baseline concurrently exhibited higher diurnal cortisol secretion than participants who slept longer. Of the sociodemographic and health-related covariates, age, sex, partnership status, and BMI significantly predicted baseline levels of diurnal cortisol volume (i.e., Level-1 intercept). As shown in Table 2, older individuals, men, participants without a partner, and participants with a higher BMI secreted significantly higher diurnal cortisol levels at baseline than younger individuals, women, participants with a partner, and participants with a lower BMI. Moreover, except for partnership status, none of the covariates significantly predicted 4-year changes in cortisol secretion (i.e., Level-1 slope). Participants who were married or cohabitating ($\beta = .58$, *SE* = .15, *T-ratio* = 3.85, $p < .01$) exhibited larger increases in their initially lower levels of cortisol secretion over 4 years as compared to their counterparts who did not have a partner ($\beta = -.18$, *SE* = .14, *T-ratio* = −1.22, $p > .10$). Finally, the analysis demonstrated

Table 2 Results of growth-curve analyses predicting 4-year changes in diurnal cortisol volume (AUC) by sociodemographic factors, baseline levels of health-related variables, and baseline levels and 2-year changes of sleep duration.

	Diurnal cortisol volume (AUC)			
	Intercept[a]		Slope	
	β (*SE*)	*T-ratio*	β (*SE*)	*T-ratio*
Level-1	12.73 (0.17)	73.89**	0.20 (0.06)	3.30**
Level-2 predictors				
Age	0.09 (0.03)	3.35**	−0.01 (0.01)	−0.98
Sex[b]	−1.43 (0.38)	−3.80**	0.19 (0.14)	1.40
Partnership status[c]	1.31 (0.35)	3.74**	−0.40 (0.13)	−2.96**
Education	0.29 (0.15)	1.89	−0.02 (0.06)	−0.34
Chronic illness	0.13 (0.12)	1.10	0.05 (0.04)	−1.21
Cortisol-related medication	0.08 (0.05)	1.41	0.01 (0.02)	0.60
BMI	0.09 (0.04)	1.94*	−0.02 (0.02)	−1.03
Smoking	1.01 (0.69)	1.46	−0.08 (0.20)	−0.40
T1 sleep duration	−0.01 (0.00)	−3.34**	0.00 (0.00)	1.22
ΔT1−T2 sleep duration	−0.03 (0.16)	−0.21	−0.18 (0.05)	−3.30**

Note. Level-1 model had 156 *dfs*. Level-2 model had 146 *dfs*.

[a] The intercept represents participants' levels of cortisol secretion at study entry, and the slope represents the within-person associations between years since study entry and cortisol secretion.

[b] Higher values represent females.

[c] Higher values represent participants without a partner.

* $p < .05$.

** $p < .01$.

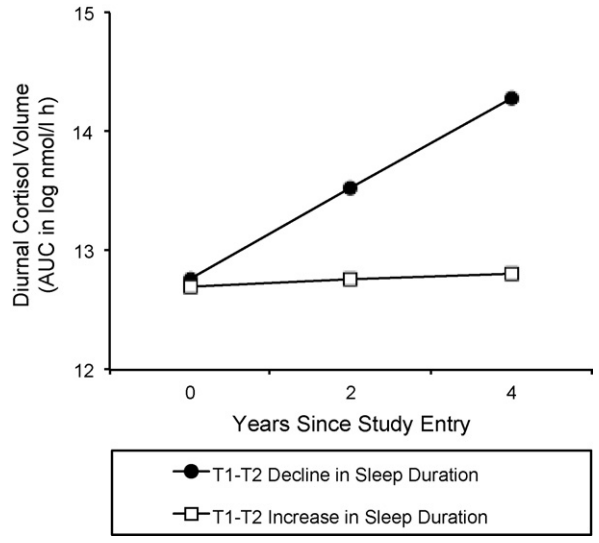

Fig. 3 Changes in cortisol secretion across 4 years, separately for participants who reported increases (+1 SD) and declines (−1 SD) in levels of sleep duration over the first 2 years of study.

that 2-year changes in sleep duration (but not baseline levels of sleep duration) significantly predicted 4-year changes in cortisol secretion (see effects on slope in Table 2).

Fig. 3 illustrates the effect of 2-year changes in sleep duration on 4-year changes in cortisol secretion by plotting the trajectories of cortisol secretion across measurements, separately for participants who exhibited increases (+1 SD) and decreases (−1 SD) in levels of sleep duration over the first 2 years of study. The observed pattern of results demonstrates that levels of cortisol secretion did not change significantly among participants who experienced increases in their sleep duration over the first 2 years of study ($\beta = 0.02$, $SE = 0.08$, $T\text{-}ratio = 0.24$, $p > .10$). By contrast, participants who reported decreases in sleep duration over the first 2 years of the study displayed steep increases in cortisol secretion over the 4-years study period ($\beta = 0.38$, $SE = 0.08$, $T\text{-}ratio = 4.68$, $p < .01$).

Predicting 4-year trajectories of sleep duration

We next reversed the previously reported growth curve analysis, and predicted in the Level-1 model the within-person variability in sleep duration (using data from T1, T2, and T3) as a function of years since study entry and a residual term. In the Level 2 model, we estimated between-person variation in the within-person intercept and slope of sleep duration as a function of the centered baseline scores of cortisol levels, 2-year changes in cortisol levels, covariates, and a random residual term.

The results of the Level-1 model showed that baseline levels of sleep duration were significantly different from zero, $\beta = 402.11$, $SE = 5.89$, $T\text{-}ratio = 68.24$, $df = 156$, $p < .01$, and increased significantly over 4 years of study, $\beta = 4.08$, $SE = 1.68$, $T\text{-}ratio = 2.43$, $df = 156$, $p < .05$. In addition, there was significant variability around the averaged Level-1 intercept and slope, χ^2s(156) > 189, $ps < .01$.

Explaining this variability, the Level-2 model showed that baseline levels of cortisol secretion, $\beta = -6.39$, $SE = 2.70$, $T\text{-}ratio = -2.36$, $df = 146$, $p < .05$, as well as 2-year changes in cortisol secretion, $\beta = -5.40$, $SE = 2.20$, $T\text{-}ratio = -2.45$, $df = 146$, $p < .05$, were negatively associated with baseline levels of sleep duration. The latter finding mirrors the results from the cross-lagged panel analyses by demonstrating that those participants who exhibited declines in cortisol secretion over 2 years reported higher baseline levels of sleep duration. Of the covariates, only sex, $\beta = -28.81$, $SE = 13.07$, $T\text{-}ratio = -2.20$, $df = 146$, $p < .05$, significantly predicted baseline levels of sleep duration (i.e., Level-1 intercept). Men reported longer sleep at baseline as compared to women. In addition, partnership status significantly predicted 4-year changes in sleep duration (i.e., Level-2 slope), $\beta = -8.39$, $SE = 3.70$, $T\text{-}ratio = -2.26$, $df = 146$, $p < .05$, revealing that participants with a partner reported larger increases in sleep duration over 4 years than their counterparts who did not have a partner. Finally, the Level-2 model also showed that neither baseline levels of cortisol secretion nor 2-year changes in cortisol secretion significantly predicted 4-year changes in participants' sleep duration.

Discussion

The study's findings demonstrate that older adults experienced increases in diurnal cortisol secretion over time, and that this effect was forecasted by individual differences in sleep duration. More specifically, results from cross-lagged panel analyses showed that lower, as compared to higher, baseline and 2-year levels of sleep duration forecasted increases in cortisol secretion over the subsequent 2 years. Further, growth-curve models substantiated these results by demonstrating that cortisol secretion generally increased over 4 years of study, and that 2-year increases in sleep duration ameliorated 4-year increases in cortisol secretion. In fact, our analyses showed a steep increase in 4-year cortisol secretion among older adults who experienced declines in their sleep duration over the first 2 years of study. By contrast, no significant changes in cortisol output were found among their counterparts who increased their sleep duration. Finally, reversed analyses showed that levels of and 2-year changes in cortisol secretion were not associated with subsequent changes in participants' sleep duration. Together, this pattern of findings supports the hypothesis that sleep patterns can have a directional, and possibly restorative, effect on older adults' diurnal cortisol secretion, and that long sleep can prevent age-normative increases in cortisol output.

It is important to note that although the cross-lagged panel analyses showed that higher baseline and 2-year levels of sleep duration ameliorated subsequent 2-year increases in cortisol secretion, the results from the growth curve model showed that baseline levels of sleep duration did not forecast 4-year changes in cortisol secretion. To this end, our analyses suggest that this pattern of findings may have emerged because some individuals exhibited changes in their sleep duration over the first 2 years of study, which were reliable predictors of 4-year changes in cortisol secretion. Thus, levels of sleep duration can be a significant predictor of older adults' cortisol changes over 2 years, and subsequent changes in cortisol secretion are more likely to emerge as a function of alterations in sleep duration during these 2 years.

These data are the first to provide direct longitudinal evidence for a mechanism that links longer sleep duration with the prevention of long-term increases in older adults' diurnal cortisol secretion. The identification of this mechanism is important because older adulthood is often associated with increasing levels of age-related challenges (e.g., health threats and loss of resources, Baltes et al., 1979; Heckhausen et al., 2010), which can elicit psychological distress and dysregulate the HPA axis (Wrosch et al., 2004, 2007). Thus, long sleep has the potential to ameliorate the adverse consequences of age-related challenges and emotional distress on increases in older adults' diurnal cortisol output, which could in turn reduce their risk of experiencing subsequent physical health declines. By contrast, short sleep could give rise to increased levels of perceived stress and negative emotions (Hamilton et al., 2007), which may dysregulate a person's HPA-axis. In addition, cortisol disturbances among older adults experiencing poor sleep could derive from their engagement in maladaptive behaviors (e.g., coffee consumption or sedentary behaviors), which they may adopt to cope with increased fatigue (e.g., Digdon and Rhodes, 2009). These conclusions are consistent with previous work theorizing that good sleep can down-regulate levels of cortisol secretion, and through this mechanism explain the observed buffering effect that restorative sleep patterns can have on the association between cortisol and subsequent physical health problems (Wrosch et al., 2008).

In addition, our research indicated that older adults experienced a fairly small, but significant, increase in their sleep duration over time (approximately 4 min each year). This finding is consistent with results from meta-analysis documenting that sleep duration typically decreases until age 60 among healthy adults, but plateaus thereafter (Ohayon et al., 2004). However, some studies have shown that sleep duration can also decline in older adulthood, and mounting evidence suggests that such effects are due to older adults' medical comorbidities rather than age per se (Foley et al., 2001; Ohayon and Vechierrini, 2002; Vitiello et al., 2002). The latter possibility further implies that sleep could become reduced over time when older adults experience an onset of severe physical health problems. Subsequently conducted analyses showed that such quadratic effects were not observed in our study, which we attribute to the relatively good health of the study participants. Nonetheless, we would expect such declines in sleep duration to emerge in later waves of our study, and suggest that this process would be likely to set in motion a downward spiral, characterized by the onset of severe health problems, reduced sleep length, increases in cortisol secretion, and subsequent physical disease.

Finally, the reported results may have some implications for clinical treatment. If restorative sleep can down-regulate increases in cortisol levels, clinical interventions should target older adults' sleep patterns (e.g., Morin et al., 1994). Such interventions could prevent the adverse consequences of age-normative challenges on the dysregulation of biological processes. Moreover, given that dysregulated cortisol patterns can forecast a variety of disease-related processes (Dekker et al., 2008; Smith et al., 2005), interventions along these lines could further contribute to the maintenance of older adults' physical health.

Limitations and future research

There are limitations to this study that need to be addressed in future research. First, we used a self-report measure to assess sleep duration. Although such self-reports of sleep duration have been validated with objective sleep assessments (Lockley et al., 1999), they could be biased by other psychological factors. In this regard, it is important to note that we analyzed changes in sleep duration over time as predictor and outcome variable, and such change measures are likely to partial out some of the potential biases found in self-reports. In addition, demonstrating that changes in sleep duration were associated with subsequent changes in cortisol output lessens the possibility that the reported findings are driven by sleep pathology. In fact, clinically significant sleeping problems could underlie individual differences in sleep duration and were not assessed in our study. However, we would expect that such sleeping problems are more likely to be associated with levels, as compared to changes, of sleep duration. In addition, we note that our sleep data were within the normative range of healthy older adults' sleep duration in the general population (Ohayon, 2004). Nonetheless, future research should include objective sleep measures and assess sleep pathology to replicate the obtained findings.

Second, our analysis focused on sleep duration and did not examine sleep efficiency (i.e., the time that individuals sleep relative to the time they spent in bed), which can also exert restorative functions (e.g., Cacioppo et al., 2002; Dew et al., 2003). In this regard, it would have been possible to incorporate a measure of sleep efficiency into our analyses. We did not pursue this possibility because the indices used for computing these variables largely overlap and sleep duration and sleep efficiency were strongly correlated in our study ($rs > .75$, $ps < .01$). However, we note here that repeating the analyses with sleep efficiency (instead of sleep duration) did not produce the same effects, and levels of, or changes in, sleep efficiency were not significantly associated with subsequent changes in cortisol output. However, given that previous work has shown that sleep efficiency can also predict health-related outcomes above and beyond sleep duration (Cohen et al., 2009), future research is needed to examine the conditions under which sleep duration and sleep efficiency predict different outcomes.

Third, we focused in our analysis on the overall volume of cortisol secretion because we reasoned that sleep duration may be particularly likely to associate with cumulative indices of cortisol output across the day. However, other research has shown that sleep duration may also be associated with cortisol slope and awakening levels of cortisol (Kumari et al., 2009). Follow-up analyses of our data showed that this was not the case for our sample, as levels of or changes in sleep duration were unrelated to changes in these alternative cortisol indices. This may imply that short sleep can influence long-term increases in cortisol secretion across the entire day, and such effects are less likely to be detected if only portions of the diurnal rhythm or changes in cortisol slope are analyzed.

Fourth, although our results were independent from a number of sociodemographic and health-related variables, some of the covariates were associated with participants' cortisol secretion. In this regard, the findings that lower

baseline cortisol levels were associated with a younger age, being female, having a partner, and a lower BMI support the validity of our measures as they reflect associations commonly found in other research (e.g., Almeida et al., 2009; Fraser et al., 1999; Kudielka et al., 1998). However, it is interesting to note that, over time, participants who had a partner exhibited larger cortisol increases, but also longer sleep, than their single counterparts. This suggests that having a partner in older adulthood can be both, a protective factor and a risk factor. In fact, such processes could derive from the possibility that although older adults often benefit from close relationships (Löckenhoff and Carstensen, 2004) and co-sleeping has been associated with reports of better sleep (Troxel, 2010), having a partner could also increase stress in the context of severe disability or becoming a caregiver (e.g., Brodaty and Hadzi-Pavlovic, 1990). While our study could not shed more light on these possibilities, we suggest that future research should conduct couple studies and track older adults' physical health, sleep, and cortisol output over time.

Finally, our analyses did not examine the complete process that could be associated with cortisol increases in old age. In this regard, our theoretical model would assume that specific age-related challenges could trigger emotional distress and dysregulate older adults' cortisol secretion (Wrosch et al., 2004, 2007), which may increase their likelihood of developing subsequent physical disease. In this cascade of events, adaptive sleep patterns and other protective factors (e.g., coping, Wrosch and Schulz, 2008) could prevent chronically high levels of psychological distress and cortisol secretion and contribute to good physical health. Thus, future studies should include a wider range of contextual, psychological, and physical health variables to illuminate how older adults can maintain adaptive biological functioning and good physical health.

Role of funding source

Preparation of this manuscript was supported by a doctoral fellowship from Concordia University to Rebecca Rueggeberg, grants and awards from the Canadian Institutes of Health Research to Carsten Wrosch and grants from the Canadian Institutes of Health Research (89736), and the Heart and Stroke Foundation of Canada awarded to Gregory Miller.

Conflict of interest

All other authors declare that they have no conflicts of interest.

Acknowledgements

We are grateful to the members of the Wrosch lab for their valuable help with data collection.

References

Aging NACO, 2006. Seniors in Canada: 2006 Report Card. National Advisory Committee.

Almeida, D.M., Piazza, J.R., Stawski, R.S., 2009. Interindividual differences and intraindividual variability in the cortisol awakening response: an examination of age and gender. Psychol. Aging 24, 819—827.

Balbo, M., Leproult, R., Van Cauter, E., 2010. Impact of sleep and its disturbances on hypothalamo—pituitary—adrenal axis activity. Int. J. Endocrinol. 1—16.

Baltes, P.B., Cornelius, S.W., Nesselroade, J.R., 1979. Cohort effects in developmental psychology. In: Nesselroade, J.R., Baltes, P.B. (Eds.), Longitudinal Research in the Study of Behavior and Development. Academic Press, New York, pp. 61—87.

Bjorntorp, P., Rosmond, R., 1999. Hypothalamic origin of the metabolic Syndrome X. Ann. N. Y. Acad. Sci. 892, 297—307.

Boscaro, M., Paoletta, A., Scarpa, E., Barzon, L., Fusaro, P., Fallo, F., Sonino, N., 1998. Age-related changes in glucocorticoid fast feedback inhibition of adrenocorticotropin in man. J. Clin. Endocrinol. Metab. 83, 1380—1383.

Brodaty, H., Hadzi-Pavlovic, D., 1990. Psychosocial effects on carers of living with persons with dementia. Aust. N. Z. J. Psychiatry 24, 351—361.

Bryk, A.S., Raudenbush, S.W., 1987. Application of hierarchical linear models to assessing change. Psychol. Bull. 101, 147—158.

Buckley, T.M., Schatzberg, A.F., 2005. On the interactions of the hypothalamic—pituitary—adrenal (HPA) axis and sleep: normal HPA axis activity and circadian rhythm, exemplary sleep disorders. J. Clin. Endocrinol. Metab. 90, 3106—3114.

Buysse, D.J., Reynolds III, C.F., Monk, T.H., Berman, S.R., Kupfer, D.J., 1989. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res. 28, 193—213.

Cacioppo, J.T., Hawkley, L.C., Berntson, G.G., Ernst, J.M., Gibbs, A.C., Stickgold, R., Hobson, J.A., 2002. Lonely days invade the nights: social modulation of sleep efficiency. Psychol. Sci. 13, 384—387.

Clements, A.D., Parker, C.R., 1998. The relationship between salivary cortisol concentrations in frozen versus mailed samples. Psychoneuroendocrinology 23, 613—616.

Cohen, S., Doyle, W.J., Alper, C.M., Janicki-Deverts, D., Turner, R.B., 2009. Sleep habits and susceptibility to the common cold. Arch. Intern. Med. 169, 62—67.

Dekker, M.J., Koper, J.W., van Aken, M.O., Pols, H.A., Hofman, A., de Jong, F.H., Kirschbaum, C., Witteman, J.C.M., Lamberts, S.W.J., Tiemeier, H., 2008. Salivary cortisol is related to atherosclerosis of carotid arteries. J. Clin. Endocrinol. Metab. 93, 3741—3747.

Dew, M.A., Hoch, C.C., Buysse, D.J., Monk, T.H., Begley, A.E., Houck, P.R., Hall, M., Kupfer, D.J., Reynolds 3rd., C.F., 2003. Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. Psychosom. Med. 65, 63—73.

Digdon, N.L., Rhodes, S., 2009. Methods used to cope with sleepiness may perpetuate sleepiness in college students with an evening type circadian preference. Biol. Rhythm Res. 40, 129—144.

Dressendörfer, R., Kirschbaum, C., Rohde, W., Stahl, F., Strasburger, C., 1992. Synthesis of a cortisol—biotin conjugate and evaluation as a tracer in an immunoassay for salivary cortisol measurement. J. Steroid Biochem. Mol. Biol. 43, 683—692.

Foley, D., Monjan, A., Masaki, K., Ross, W., Havlik, R., White, L., Launer, L., 2001. Daytime sleepiness is associated with 3-year incident dementia and cognitive decline in older Japanese—American men. J. Am. Geriatr. Soc. 49, 1628—1632.

Fraser, R., Ingram, M.C., Anderson, N.H., Morrison, C., Davies, M., Connell, J.M.C., 1999. Cortisol effects on body mass, blood pressure, and cholesterol in the general population. Hypertension 33, 1364—1368.

Gangwisch, J.E., Heymsfield, S.B., Boden-Albala, B., Buijs, R.M., Kreier, F., Pickering, T.G., Rundle, A.G., Zammit, G.K., Malaspina, D., 2006. Short sleep duration as a risk factor for hypertension:

analyses of the first National Health and Nutrition Examination Survey. Hypertension 47, 833—839.
Greenspan, S.L., Rowe, J.W., Maitland, L.A., McAloon-Dyke, M., Elahi, D., 1993. The pituitary—adrenal glucocorticoid response is altered by gender and disease. J. Gerontol. 48A, M72—M77.
Hamilton, N.A., Nelson, C., Stevens, N., Kitzman, H., 2007. Sleep and psychological well-being. Soc. Indic. Res. 82, 147—163.
Heckhausen, J., Wrosch, C., Schulz, R., 2010. A motivational theory of life-span development. Psychol. Rev. 117, 32—60.
Heim, C., Ehlert, U., Hellhammer, D.H., 2000. The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. Psychoneuroendocrinology 25, 1—35.
Kern, W., Dodt, C., Born, J., Fehm, H.L., 1996. Changes in cortisol and growth hormone secretion during nocturnal sleep in the course of aging. J. Gerontol. 51A, M3—M9.
Kudielka, B.M., Hellhammer, J., Hellhammer, D.H., Wolf, O.T., Pirke, K.M., Varadi, E., et al., 1998. Sex differences in endocrine and psychological responses to psychosocial stress in healthy elderly subjects and the impact of a 2-week dehydropiandrosterone treatment. J. Clin. Endocrinol. Metab. 83, 1756—1761.
Kumari, M., Badrick, E., Ferrie, J., Perski, A., Marmot, M., Chandola, T., 2009. Self-reported sleep duration and sleep disturbance are independently associated with cortisol secretion in the Whitehall II Study. J. Clin. Endocrinol. Metab. 94, 4801—4809.
Leproult, R., Copinschi, G., Buxton, O., Van Cauter, E., 1997. Sleep loss results in an elevation of cortisol levels the next evening. Sleep 10, 865—870.
Löckenhoff, C.E., Carstensen, L.L., 2004. Socioemotional selectivity theory, aging, and health: the increasingly delicate balance between regulating emotions and making tough choices. J. Pers. 72, 1395—1424.
Lockley, S.W., Skene, D.J., Arendt, J., 1999. Comparison between subjective and actigraphic measurement of sleep and sleep rhythms. J. Sleep Res. 8, 175—183.
Lupien, S.J., de Leon, M., De Santi, S., Convit, A., Tarshish, C., Nair, N.P.V., Thakur, M., McEwen, B.S., Hauger, R.L., Meaney, M.J., 1998. Cortisol levels during human aging predict hippocampal atrophy and memory deficits. Nat. Neurosci. 1, 69—73.
Lupien, S.J., Schwartz, G., Ng, Y.K., Fiocco, A., Wan, N., Pruessner, J.C., Meaney, M.J., Nair, N.P., 2005. The Douglas Hospital longitudinal study of normal and pathological aging: summary of findings. J. Psychiatry Neurosci. 30, 328—334.
Lupien, S.J., McEwen, B.S., Gunnar, M.R., Heim, C., 2009. Effects of stress throughout the lifespan on the brain, behaviour and cognition. Nat. Rev. Neurosci. 10, 434—445.
McEwen, B.S., Sapolsky, R.M., 1995. Stress and cognitive function. Curr. Opin. Neurobiol. 5, 205—216.
McEwen, B.S., Stellar, E., 1993. Stress and the individual: mechanisms leading to disease. Arch. Intern. Med. 153, 2093—2101.
Miller, G.E., Chen, E., Zhou, E.S., 2007. If it goes up, must it come down? Chronic stress and the hypothalamic—pituitary—adrenocortical axis in humans. Psychol. Bull. 133, 25—45.
Morin, C.M., Culbert, J.P., Schwartz, S.M., 1994. Nonpharmacological interventions for insomnia: a meta-analysis of treatment efficacy. Am. J. Psychiatry 151, 1172—1180.
Ohayon, M.M., 2004. Interactions between sleep normative data and sociocultural characteristics in the elderly. J. Psychosom. Res. 56, 479—486.
Ohayon, M.M., Vechierrini, M.F., 2002. Daytime sleepiness is an independent predictive factor for cognitive impairment in the elderly population. Arch. Intern. Med. 162, 201—208.
Ohayon, M.M., Carskadon, M.A., Guilleminault, C., Vitiello, M.V., 2004. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. Sleep 27, 1255—1273.
Otte, C., Hart, S., Neylan, T.C., Marmar, C.R., Yaffe, K., Mohr, D.C., 2005. A meta-analysis of cortisol response to challenge in human aging: importance of gender. Psychoneuroendocrinology 30, 80—91.
Patel, S.R., Malhotra, A., White, D.P., Gottlieb, D.J., Hu, F.B., 2006. Association between reduced sleep and weight gain in women. Am. J. Epidemiol. 164, 947—954.
Raison, C.L., Miller, A.H., 2003. When not enough is too much: the role of insufficient glucocorticoid signaling in the pathophysiology of stress-related disorders. Am. J. Psychiatry 160, 1554—1565.
Raudenbush, S.W., Bryk, A.S., Cheong, Y.K., Congdon, R.T., 2004. HLM: Version 6. Scientific Software International, Chicago.
Rodenbeck, A., Huether, G., Ruther, E., Hajak, G., 2002. Interactions between evening and nocturnal cortisol secretion and sleep parameters in patients with severe chronic primary insomnia. Neurosci. Lett. 324, 159—163.
Rotermann, M., 2006. Seniors' health care use. Health Reports Suppl., 8. Statistics Canada, Catalogue 82-003, pp. 33—45.
Sapolsky, R.M., 1992. Stress, the Aging Brain, and the Mechanisms of Neuron Death. MIT Press, Cambridge, MA.
Sapolsky, R.M., Romero, L.M., Munck, A.U., 2000. How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocr. Rev. 21, 55—89.
Schaeffer, M.A., Baum, A., 1984. Adrenal cortical response to stress at Three Mile Island. Psychosom. Med. 46, 227—237.
Seeman, T.E., Robbins, R.J., 1994. Aging and hypothalamic—pituitary—adrenal response to challenge in humans. Endocr. Rev. 15, 233—260.
Seeman, T.E., McEwen, B.S., Singer, B.H., Albert, M.S., Rowe, J.W., 1997. Increase in urinary cortisol excretion and memory declines: MacArthur studies of successful aging. J. Clin. Endocrinol. Metab. 82, 2458—2465.
Shaver, J.L.F., Johnston, S.K., Lentz, M.J., Landis, C.A., 2002. Stress exposure, psychological distress, and physiological stress activation in midlife women with insomnia. Psychosom. Med. 64, 793—802.
Smith, G.D., Ben, S.Y., Beswick, A., Yarnell, J., Lightman, S., Elwood, P., 2005. Cortisol, testosterone, and coronary heart disease: prospective evidence from the Caerphilly study. Circulation 112, 332—340.
Späth-Schwalbe, E., Scholler, T., Kern, W., Fehm, H.L., Born, J., 1992. Nocturnal adrenocorticotropin and cortisol secretion depends on sleep duration and decreases in association with spontaneous awakening in the morning. J. Clin. Endocrinol. Metab. 75, 1431—1435.
Spiegel, K., Leproult, R., Van Cauter, E., 1999. Impact of sleep debt on metabolic and endocrine function. Lancet 354, 1435—1439.
Steiger, A., 2003. Sleep and endocrinology. J. Intern. Med. 254, 13—22.
Troxel, W.M., 2010. It's more than sex: exploring the dyadic nature of sleep and implications for health. Psychosom. Med. 72, 578—586.
Van Cauter, E., Turek, F.W., 1994. Endocrine and other biological rhythms. In: DeGroot, L.J. (Ed.), Endocrinology. W.B. Saunders, Philadelphia, pp. 2487—2548.
Van Cauter, E., Leproult, R., Kupfer, D.J., 1996. Effects of gender and age on the levels and circadian rhythmicity of plasma cortisol. J. Clin. Endocrinol. Metab. 81, 2468—2473.
Van Cauter, E., Leproult, R., Plat, L., 2000. Age-related changes in slow wave sleep and REM sleep and relationship with growth hormone and cortisol levels in healthy men. JAMA 284, 861—868.
Vitiello, M.V., Moe, K.E., Prinz, P.N., 2002. Sleep complaints cosegregate with illness in older adults: clinical research informed by

and informing epidemiological studies of sleep. J. Psychosom. Res. 53, 555—559.
Weibel, L., Follenius, M., Spiegel, K., Ehrhart, J., Brandenberger, G., 1995. Comparative effect of night and daytime sleep on the 24-hour cortisol secretory profile. Sleep 7, 549—556.
Weiner, H., 1992. Perturbing the Organism: The Biology of Stressful Experience. University of Chicago Press, Chicago.
Weitzman, E.D., Zimmerman, J.C., Czeisler, C.A., Ronda, J., 1983. Cortisol secretion is inhibited during sleep in normal man. J. Clin. Endocrinol. Metab. 2, 352—358.
Wilkinson, C.W., Peskind, E.R., Raskind, M.A., 1997. Decreased hypothalamic—pituitary—adrenal axis sensitivity to cortisol feedback inhibition in human aging. Neuroendocrinology 65, 79—90.
Wrosch, C., Schulz, R., 2008. Health engagement control strategies and 2-year changes in older adults' physical health. Psychol. Sci. 19, 537—541.
Wrosch, C., Schulz, R., Heckhausen, J., 2004. Health stresses and depressive. Symptomatology in the elderly: a control-process approach. Curr. Direct. Psychol. Sci. 13, 17—20.
Wrosch, C., Schulz, R., Miller, G.E., Lupien, S., Dunne, E., 2007. Physical health problems, depressive mood, and cortisol secretion in old age: buffer effects of health engagement control strategies. Health Psychol. 26, 341—349.
Wrosch, C., Miller, G.E., Lupien, S., Pruessner, J.C., 2008. Diurnal cortisol secretion and 2-year changes in older adults' acute physical symptoms: the moderating roles of negative affect and sleep. Health Psychol. 27, 685—693.
Wrosch, C., Miller, G.E., Schulz, R., 2009. Cortisol secretion and functional disabilities in old age: importance of using adaptive control strategies. Psychosom. Med. 71, 996—1003.