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Childhood Sleep Duration and Lifelong Mortality Risk

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Objective: Sleep duration is known to significantly affect health in adults and children, but little is understood about long-term associations. This prospective cohort study is the first to examine whether childhood sleep duration is associated with lifelong mortality risk. ***Method:*** Data from childhood were refined and mortality data collected for 1,145 participants from the Terman Life Cycle Study. Participants were born between 1904 and 1915, lived to at least 1940, and had complete age, bedtime, and waketime data at initial data collection (1917–1926). Homogeneity of the cohort sample (intelligent, mostly White) limits generality but provides natural control of common confounds. Through 2009, 1,039 participants had confirmed deaths. Sleep duration was calculated as the difference between each child's bed and wake times. Age-adjusted sleep (deviation from that predicted by age) was computed. Cox proportional hazards survival models evaluated childhood sleep duration as a predictor of mortality separately by sex, controlling for baseline age. ***Results:*** For males, a quadratic relation emerged: Male children who underslept or overslept compared with peers were at increased risk of lifelong all-cause mortality (HR = 1.15, CIs [1.05, 1.27]). Effect sizes were smaller and nonsignificant in females (HR = 1.02, CIs [0.91, 1.14]). ***Conclusions:*** Male children with shorter or longer sleep durations than expected for their age were at increased risk of death at any given age in adulthood. The findings suggest that sleep may be a core biobehavioral trait, with implications for new models of sleep and health throughout the entire life span.

Keywords: sleep, mortality, life span, childhood, development

Deviation from normative sleep patterns is an important, well-documented predictor of health and well-being. In adults, sleep problems are associated with depression, anxiety, and hostility (Pilcher, Ginter, & Sadowsky, 1997; Roberts, Shema, Kaplan, & Strawbridge, 2000; Sbarra & Allen, 2009), more problems at work (Kuppermann et al., 1991), loneliness and a lack of social support (Åkerstedt et al., 2002; Cacioppo et al., 2002a; Cacioppo et al., 2002b), fatigue (Kuppermann et al., 1991; Pilcher et al., 1997), a greater probability of contracting the common cold (Cohen et al., 2008; Cohen, Doyle, Alper, Janicki-Deverts, & Turner, 2009), and poor physical health (Kuppermann et al., 1991; Pilcher et al., 1997). Sleep duration is associated with obesity (Cappuccio et al., 2008), diabetes (Spiegel, Knutson, Leproult, Tasali, & Van Cauter, 2005), hypertension (Cappuccio et al., 2007), and cardiovascular disease (Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011). However, little is understood about the causal interrelationships, such as whether improvements in sleep will directly improve health and well-being, whether sleep impairments are primarily a result of disruptions in physical or mental health, or whether sleep and health and well-being are correlated because of biopsychosocial third variables that are associated with each. The present study begins to untangle these relationships using a very long-term data set.

Strikingly, sleep is a good predictor of mortality risk (Cappuccio, D'Elia, Strazzullo, & Miller, 2010; Gallicchio & Kalesan, 2009; Hammond, 1964; Kripke, Simons, Garfinkel, & Hammond, 1979), which is arguably the best single measure of overall health (Friedman & Kern, 2014). In the first significant analysis, Hammond (1964) found that adult men who reported sleeping about 7 hours per night had the lowest risk. Following this, Kripke, Simons, Garfinkel, and Hammond (1979) found that individuals sleeping less than 4 hr or more than 10 hr were at increased mortality risk, with people with long sleep durations at relatively greater risk than people with short sleep durations. These mortality associations were confirmed in two recent meta-analyses of prospective cohort studies (Cappuccio et al., 2010; Gallicchio & Kalesan, 2009). Short sleep duration was associated with significantly greater risk of all-cause mortality (death due to any cause),

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but not specific causes of death. Long sleep duration was associated with significantly greater risk of death due to all causes, as well as cardiovascular disease and cancer individually (Gallicchio & Kalesan, 2009). Considering moderators of these associations, for short sleep, the effect did not change depending on age, gender, adjustment for socioeconomic status, or variations in the definition of short sleep (Cappuccio et al., 2010). For long sleep, the effect was stronger in older cohorts and increased as the definition of long duration sleep became more and more extreme (Cappuccio et al., 2010). Overall, the two meta-analyses confirmed a U-shaped association between sleep and mortality risk, such that people with especially short and long sleep durations are at increased risk of dying, with long sleep conferring greater risk than short sleep, and risk increasing with greater deviation from the study reference group.

Given the associations of sleep duration with the host of psychological, social, health behavioral, and disease variables noted above, long-term or even life span models may be relevant to fully understanding sleep and mortality risk. Prospective studies of sleep in healthy children can shed light on the issue of whether sleep duration may be associated with mortality risk because it is connected with chronic unhealthy trajectories, and whether sleep may be an early marker for health problems that will develop later in life. Such very long-term studies may also provide hints about how relevant health processes unfold across the decades.

In 1913, the Stanford psychologist Lewis Terman presciently asked, "What is the optimum amount of sleep for physical and mental efficiency, and how are we affected by variations above or below this amount?" (Terman & Hocking, 1913, p. 138). Now, 100 years after Terman asked this very important question, relatively little research has been conducted on this topic using a long-term or life course focus. In cross-sectional data from a national random sample of American elementary school-age children and adolescents, "inadequate sleep" as reported by parents was correlated with poorer health and higher depressive symptoms (Smaldone, Honig, & Byrne, 2007). Longitudinally, Snell, Adam, and Duncan (2007) found that short sleep duration was associated with higher body mass index (BMI) 5 years after baseline, controlling for demographic variables, parent income, parent education, and body mass index at baseline. This relationship was confirmed in the Dunedin longitudinal study (Landhuis, Poulton, Welch, & Hancox, 2008), in which children with short sleep durations again had significantly higher BMIs at 32 years of age, possibly indicative of a metabolic disturbance. In a meta-analysis, the pooled odds ratio for short sleep duration and obesity in children was 1.89 (Cappuccio et al., 2008). It is unknown whether, when, or why these associations to unhealthy outcomes might extend beyond young adulthood, and whether other unusual childhood sleep profiles (e.g., long sleeping) might be associated with health risks in later years.

The current study explores relations between *childhood* sleep duration and *lifelong* mortality risk in a sample of healthy children followed from childhood throughout their entire lives. Fittingly, we use Terman's sleep data collected on children and adolescents to address this question. Results from the current study begin to elucidate the extent to which associations between sleep and health extend throughout the entire life course.

Method

Data Collection

Data were refined and supplemented with death certificates to ascertain and verify length of life for participants from the Terman Life Cycle study, started by Lewis Terman between 1917 and 1921. All archival data supplementation and death certificate collection procedures were reviewed and approved by the University of California, Riverside Institutional Review Board. Terman asked teachers from California schools to nominate the youngest and brightest children in their classes, and those with an IQ of at least 135 were admitted to the study (Terman, 1925). Terman added participants until 1928, yielding a final sample of 1,528 intelligent boys ($N = 856$) and girls ($N = 672$). Participants were mostly White, middle-class children who were then followed every 5 to 10 years throughout their lives, with survey assessments through 1999. In order to be consistent with previous work using these data, the current study was limited to participants born between 1904 and 1915. Importantly, to be conservative and eliminate those who developed fatal illnesses by young adulthood, participants who died prior to 1940 were excluded from all analyses. Of the original sample, 1,373 participants were born between 1904 and 1915, 50 died prior to 1940, and an additional 178 did not have complete childhood sleep information, yielding a final sample size of 1,145 (633 M, 512 F).

Analyses by Sears (1979) show that the minimal attrition that occurred did not significantly change the makeup of the group with respect to a variety of demographic characteristics, including age, income, and education. We too find no differential attrition in that various survival analyses do not predict who dropped out, and lifetime attrition of those with sleep data was only 9.3%, as most death certificates have been located. Other research confirms that participants in the Terman sample are similar to other bright, middle-class cohorts and might most profitably be regarded as derived from an important and productive segment of 20th century White U.S. society (Sears, 1984; Subotnik, Karp, & Morgan, 1989). The sample has been used successfully to uncover surprising but highly replicable findings about psychosocial predictors of health and longevity across the decades (Friedman & Martin, 2011; Kern & Friedman, 2008). Although not directly generalizable to the U.S. population, the relatively homogeneous nature of the sample does not unduly restrict the range (of variation) on either sleep or mortality risk (see Results section).

This was initially a healthy sample. Compared with an unselected control group, children in this study had better diets and were less likely to suffer from headaches or symptoms of general weakness.[1] Notably, "marked" or "extreme" mouth breathing (a sign of possible sleep apnea) was reported one third as frequently in this sample as in the general school population (Terman, 1925). Many of the children were given complete medical exams by a physician, and Terman reported "the examining physicians are in accord in the belief that on the whole the children of this group are physically superior to unselected children of corresponding age in the school population" (Terman, 1925, p. 251). Between the good health of this sample and the elimination from analysis of all who

[1] Unfortunately, data from this unselected control group from the 1920s are not available.

died before 1940, there is little chance that significant life-threatening comorbid chronic illness in childhood is confounded with the sleep measures. Indeed, the associations reported here represent a conservative test of sleep-mortality associations.

Sleep Duration and Age-Adjusted Sleep

Sleep duration was measured at a single time point using a baseline questionnaire filled out upon each participant's entry into the study. About 99% of the time, the information was reported by an adult family member (96% of the time, the parent or parents). Respondents answered: "Usual hour of going to sleep? Of waking?" For bedtime, a total of 1,178 parents or participants responded, with 1,168 reporting a regular bedtime. For waketime, a total of 1,160 parents or participants responded, with 1,152 reporting a regular wake-time. To indicate typical sleep duration, the difference between the participant's bedtime and waketime was calculated in hours. Table 1 summarizes reported bedtimes, waketimes, and calculated sleep duration frequencies.

Optimal sleep duration differs for children versus adolescents. Because an 8-year old who sleeps 7 hr may have very different outcomes than a 16-year-old who sleeps 7 hr, it was important to adjust for age. Age was calculated by taking the difference between each participant's birthdate and the date the baseline survey was completed (M age = 11.88 years, SD = 2.86; range, 5–21 years).[2] Then to obtain variations from age-expected sleep durations, reported sleep duration was regressed on age, yielding the following regression equation ($R^2 = .38$, $p < .0001$):[3]

$$Sleep\ Duration = 12.17 - 0.17 * age$$

Thus, each participant's residual value represents the extent to which the participant slept longer or shorter than what is predicted for their age. For example, a residual of +1.0 indicates that the participant slept exactly 1 hr more than what would be predicted for his or her age, whereas a residual of −1.0 indicates that the participant slept 1 hr less than what would be predicted for his or her age. Note that comparisons were made within the sample, rather than from population norms. Most sleep recommendations (from the government or medical groups) are not evidence-based, but rather are simply descriptive statistics of how much a given population is sleeping. As summarized in Table 2, the predicted amounts of sleep for each age group in the Terman sample mapped well onto current CDC sleep recommendations (Centers for Disease Control & Prevention, 2010) as well as descriptive sleep data Terman collected in 1913 on an earlier, unselected sample (Terman & Hocking, 1913).

Statistical Analyses

A series of Cox proportional hazards regression models, a form of survival analysis, were fitted using SAS® software, version 9.3 (SAS Institute, Cary, NC). Hazard regression analysis simultaneously considers the mortality rate at all ages across time and how this rate is related to the predictor. Hazard regression models properly treat censored (or unobserved) data, including left-censored data (created by our decision to limit analyses to survival to 1940 and beyond) and right-censored data (where age at death is unknown). Survival analysis is advantageous in this situation because rather than discarding cases who have not yet died or who were lost to follow-up during the study, the participants are censored at their last age of follow-up (Cox, 1972).

In Model I (age only), age was used to predict mortality risk. To aid in the interpretability of the parameter estimates, age was centered at age 12, such that the hazard ratio indicates the risk of death for each year of age, compared with a 12-year-old. In Model II (linear model), centered age and age-adjusted sleep were used to predict mortality risk. Age-adjusted sleep was centered on the sleep duration predicted by the regression equation, such that the hazard ratio indicates risk for deviations from the amount of sleep expected for his or her age. In Model III (curvilinear model or U-shaped model), centered age, age-adjusted sleep, and squared age-adjusted sleep were used to predict risk, to determine whether extreme (squared) sleep values in either direction (low or high) are associated with greater mortality risk. Sleep duration was again centered on the values from the regression equation. As models were nested within each other, deviance statistics across models were compared using chi-square difference tests to establish the significance of added parameters. There was no evidence of nonproportionality (an assumption of the Cox models).

We first examined the associations between sleep and mortality for the full sample with sex as a covariate. Given commonly reported major gender differences in health and longevity outcomes, it was unsurprising that sex emerged as a significant predictor of mortality, even controlling for childhood sleep (results for the full sample are as follows: age $HR = 0.954$, $p < .0001$; sex $HR = 0.764$, $p < .0001$; age-adjusted sleep $HR = 1.067$, $p = .13$; squared age-adjusted sleep $HR = 1.085$, $p = .03$). Thus, we report analyses that were conducted separately by sex. Finally, a series of supplemental analyses were performed to determine whether results may have been a function of outliers or confounding individual difference factors in childhood.

Control Variables

Childhood personality and health variables reported at the start of the study were examined as control variables in follow-up survival analyses. Childhood personality traits (reported by parents and teachers) were included because they are known predictors of mortality in these data (Friedman et al., 1993): Conscientiousness ($\alpha = .76$; prudence-forethought, freedom from vanity-egotism, conscientiousness, truthfulness); Cheerfulness ($\alpha = .52$; sense of humor and cheerfulness-optimism); Permanency of Moods (single item reflecting emotional stability); Energy ($\alpha = .43$; physical energy, preference for games requiring lots of exercise, preference for playing outdoors), Further, due to their conceptual relevance here, we also examined: parent-reported health of child (using a 5-point Likert scale ranging from *poor or very poor* to *very good*),

[2] Most participants (81%) filled out the questionnaire in 1922, but some were collected as early as 1917 and others as late as 1926. Participants who returned questionnaires from 1917 to 1920 missing the exact year (N = 27) were coded as returning the questionnaire in 1920. In addition, 40 participants were missing data on the month they filled out the survey, so the median month (May) was used. As results do not change if these individuals are eliminated, results are reported for the full sample.

[3] A quadratic model did not improve model fit, and results did not change when sleep duration was regressed on age separately for males and females. Thus, the results reported here are based on the residuals for the linear regression of sleep on age for the full sample.

Table 1
*Frequency of Bedtimes, Waketimes, and Sleep Duration (*N*, %)*

Time	Full sample	Male	Female
Bedtime			
6:00 p.m.–6:59 p.m.	2 (0.17)	0 (0)	2 (0.37)
7:00 p.m.–7:59 p.m.	85 (7.01)	40 (5.91)	45 (8.41)
8:00 p.m.–8:59 p.m.	485 (40.02)	269 (39.73)	216 (40.37)
9:00 p.m.–9:59 p.m.	484 (39.93)	270 (39.88)	214 (40.00)
10:00 p.m.–10:59 p.m.	103 (8.50)	62 (9.16)	41 (7.66)
11:00 p.m.–11:59 p.m.	9 (0.74)	7 (1.03)	2 (0.37)
After midnight, late*	2 (0.17)	2 (0.30)	0 (0)
Varies widely*	8 (0.66)	4 (0.59)	4 (0.75)
Waketime			
5:00 a.m.–5:59 a.m.	12 (0.99)	7 (1.03)	5 (0.93)
6:00 a.m.–6:59 a.m.	411 (33.91)	227 (33.53)	184 (34.39)
7:00 a.m.–7:59 a.m.	693 (57.18)	378 (55.83)	315 (58.88)
8:00 a.m.–8:59 a.m.	36 (2.97)	24 (3.55)	12 (2.24)
Varies widely, no habitual time*	7 (0.58)	6 (0.89)	1 (0.19)
Late*	1 (0.08)	1 (0.15)	0 (0)
Sleep duration			
7 hr	3 (0.26)	3 (0.47)	0 (0)
8 hr	34 (2.97)	18 (2.84)	16 (3.13)
9 hr	217 (18.95)	123 (19.43)	94 (18.36)
10 hr	521 (45.50)	300 (47.39)	221 (43.16)
11 hr	311 (27.16)	156 (24.64)	155 (30.27)
12 hr	58 (5.07)	33 (5.21)	25 (4.88)
13 hr	1 (0.09)	0 (0)	1 (0.20)
Average (*SD*)	10.12 (0.89)	10.09 (0.89)	10.16 (0.89)

Note. * These categories were not used in calculating sleep duration. The average sleep duration for men and women were not significantly different from each other, $t(1143) = 1.41$, $p = .16$.

SES ($\alpha = .90$; standardized and summed parental education and occupation levels), and IQ (overall best-estimated IQ level determined by Terman). For more details, see Friedman et al. (1993), Schwartz et al. (1995), and Kern and Friedman (2009).

Table 2
Comparisons of Predicted Sleep, Norms, and Sleep Recommendations, in Hours

Age, in years	This sample (*N*)	Terman & Hocking, 1913	CDC, 2010
5	11.32 (1)	—	10–13
6	11.15 (29)	11.23	10–11
7	10.98 (52)	10.68	10–11
8	10.81 (116)	10.70	10–11
9	10.64 (133)	10.22	10–11
10	10.47 (140)	9.93	8.5–11
11	10.30 (157)	10.00	8.5–9.5
12	10.13 (141)	9.60	8.5–9.5
13	9.96 (112)	9.52	8.5–9.5
14	9.79 (89)	9.10	8.5–9.5
15	9.62 (55)	8.90	8.5–9.5
16	9.45 (51)	8.50	8.5–9.5
17	9.28 (54)	8.77	8.5–9.5
18	9.11 (15)	8.77	7–9

Note. Estimates from the current sample are the amount of sleep predicted based on age using regression; data from Terman and Hocking (1913), are norms from a similar time period; data from CDC (2010) are modern sleep recommendations.

Results

Survival Analyses

Table 3 presents model results for males. In Model I, there was a linear relationship between centered age and mortality (*HR* = 0.96, 95% CIs [0.94, 0.99]), in which older males at baseline were at slightly lower risk of mortality than younger males. In Model II, age-adjusted sleep was added as a linear predictor of mortality risk. Sleep was not a significant predictor; boys with longer sleep durations relative to their same-age peers were at nonsignificantly greater risk than boys with shorter sleep durations (*HR* = 1.07, CIs [0.95, 1.21]). Deviance statistics indicated that Model II did not fit significantly better than Model I ($\Delta\chi^2 = 1.41$, $p = .23$). In Model III, squared age-adjusted sleep was added as a quadratic predictor of mortality risk. This model fit significantly better than both Model II and Model I. Specifically, there was a significant quadratic but not linear effect. That is, boys with higher age-adjusted sleep scores were not at greater risk than boys with lower age-adjusted sleep scores (*HR* = 1.10, CIs [0.98, 1.23]), whereas boys with unusual or extreme sleep patterns (i.e., sleeping more or less than peers in either direction) were at the greatest risk (*HR* = 1.15, CIs [1.05, 1.27]).

Table 4 reports model results for females. In Model I, centered age significantly related to lower mortality risk (*HR* = 0.94, CIs [0.91, 0.98]); older females at baseline were at lower mortality risk than younger females. In Model II, girls with longer sleep durations relative to their same-age peers were at slightly greater risk than girls with shorter sleep durations (*HR* = 1.03, CIs [0.91,

Table 3
Results of Fitting Three Cox Regression Models to the Sleep Data for Males (N = *633*)

Males	Model I	Model II	Model III
Parameter estimates, standard errors, and *p* values			
Centered age	−0.04 *SE* = 0.01 *p* = .01	−0.04 *SE* = 0.01 *p* = .008	−0.04 *SE* = 0.01 *p* = .01
Age-adjusted sleep		0.07 *SE* = 0.06 *p* = .24	0.09 *SE* = 0.06 *p* = .10
Squared age-adjusted sleep			0.14 *SE* = 0.05 *p* = .005
Hazard ratios and 95% confidence intervals			
Centered age	0.96 [0.94, 0.99]	0.96 [0.94, 0.99]	0.96 [0.94, 0.99]
Age-adjusted sleep		1.07 [0.95, 1.21]	1.10 [0.98, 1.23]
Squared age-adjusted sleep			1.15 [1.05, 1.27]
Goodness-of-fit			
−2LL	6445.168	6443.756	6436.835
AIC	6447.168	6447.756	6442.835
SBC	6451.551	6456.523	6455.986
Model comparisons	Model I	Model II	Model III
Model I	—	$\chi^2(1) = 1.412$ *p* = .23	$\chi^2(2) = 8.333$ *p* = .02
Model II	—	—	$\chi^2(1) = 6.921$ *p* = .009

1.18]), but this difference was not statistically significant. In line with these results, deviance statistics indicated that Model II did not fit significantly better than Model I ($\Delta\chi^2 = .27$, $p = .60$). In Model III, neither linear nor quadratic sleep effects were significant, and this model did not fit significantly better than Model I or Model II.

We conducted additional analyses to capture the magnitude of this quadratic effect. We used Kaplan Meier estimates to examine the median age of death for male participants deviating less than 20% and more than 80% from the average amount of sleep expected for their age, using the estimates from Model III. Male participants in the lowest quintile (20th percentile) on squared age-adjusted sleep [that is, those who slept within 0.038 squared hr (± 12 min) of their peers, $N = 110$)] died at a median age of 80. Males who were above the 80th percentile, sleeping within .86 to 9.12 squared hr (± 56 to 181 min) of their peers ($N = 128$), died at a median age of 78. A longevity difference of this magnitude is quite important in survival studies of this sort, comparable with other important effects such as blood pressure (cf. Friedman et al., 1993; Meyer et al., 2001).

To further illustrate differences in mortality risk, we calculated risk scores for a variety of child ages and sleep residuals (see Table 5 and Figure 1). Risk scores summarize the effect of several predictors simultaneously and are predicted values based on these particular models of sleep and mortality in the Terman data. These risk scores assess the relative hazard of participants with various combinations of ages and sleep durations, relative to an individual in the baseline hazard function (e.g., 12-year-old children sleeping what is predicted for their age; Singer & Willett, 2003). In these data, 12-year-old male participants sleeping 180 min more than their peers were at 4.77 times the mortality risk of those sleeping the same amount as their peers; 12-year-old male participants sleeping 180 min less than their peers were at 2.71 times the mortality risk. This association is visually presented in Figure 1. Risk scores are given in Table 5 for females from the fullest model fitted (Model III) for comparison, although sleep duration results did not significantly predict mortality risk for females.

Supplemental Analyses and Checks

To examine the robustness of these associations, we conducted a series of supplemental analyses. Childhood sleep predicts mortality risk for boys, but can this association be readily explained by associations between childhood sleep and other individual characteristics? First, we examined whether individual characteristics explain the association between childhood sleep and mortality risk for boys. We tested the survival model, including childhood conscientiousness, permanency of mood, and cheerfulness, known personality predictors of mortality in these data (Friedman et al., 1993). Further, we examined energy, parent-reported health of child, SES, and IQ (cf. Schwartz et al., 1995). Analyses showed that conscientiousness, health, energy, and IQ were significantly correlated with sleep at the bivariate level, but the relationship between squared age-adjusted sleep and mortality was not reduced by including them in the model. The association was remarkably robust, and including these variables the model only reduced the association from $HR = 1.145$, $p = .007$, to HR 1.141, $p = .01$ in these follow-up analyses.

Table 4
*Results of Fitting Three Cox Regression Models to the Sleep Data for Females (*N = *512)*

	Model I	Model II	Model III
Parameter estimates, standard errors, and *p* values			
Centered age	−0.06 *SE* = 0.02 *p* < .001	−0.06 *SE* = 0.02 *p* < .001	−0.06 *SE* = 0.02 *p* < .001
Age-adjusted sleep		0.03 *SE* = 0.07 *p* = .60	0.04 *SE* = 0.07 *p* = .58
Squared age-adjusted sleep			0.02 *SE* = 0.06 *p* = .78
Hazard ratios and 95% confidence intervals			
Centered age	0.94 [0.91, 0.98]	0.94 [0.91, 0.98]	0.94 [0.91, 0.98]
Age-adjusted sleep		1.03 [0.91, 1.18]	1.04 [0.91, 1.18]
Squared age-adjusted sleep			1.02 [0.91, 1.14]
Goodness-of-fit			
−2LL	4625.068	4624.799	4624.720
AIC	4627.068	4628.799	4630.720
SBC	4631.170	4637.004	4643.028
Model comparisons	Model I	Model II	Model III
Model I	—	$\chi^2(1) = 0.269$ *p* = .60	$\chi^2(2) = 0.348$ *p* = .84
Model II	—	—	$\chi^2(1) = 0.079$ *p* = .78

Next, we examined residuals and outliers. Examination of both deviance and score residuals (Singer & Willett, 2003) suggested that the model fit equally well for extreme sleepers and average sleepers. Moreover, dropping outlying cases that exceeded ± 2.5 standard deviations on the score residuals on age-adjusted sleep ($N = 7$ cases) and squared age-adjusted sleep ($N = 8$ cases) did not decrease the effect size or significance of the parameters, and indeed strengthened the effect sizes (i.e., when eliminating the 11 cases that had excessively large score residuals on either age-adjusted sleep or squared age-adjusted sleep, the hazard ratio for age-adjusted sleep increased to 1.20, CIs [1.07, 1.36]).

We have previously applied the Pearson-Aitken-Lawley correction (which examines influence of restricted range of a third variable) to check for possible IQ selection effects on health-relevant traits; the narrowed IQ range produces little if any distortion on health-related variables in terms of means, variances, or covariances (Reynolds, McArdle, Kern, & Friedman, 2013). Results of this analysis applied to sleep variables showed that selection effects of IQ and education on the sleep variables were negligible with respect to means ($d < .22$) and variances (0% change), and therefore could not influence the results of the present study. Overall, the results of the final model for males are extraordinarily robust to a variety of diagnostic techniques.

Finally, to explore potential lifelong biopsychosocial pathways linking childhood sleep duration with lifelong mortality risk, we examined associations between childhood sleep durations and specific causes of death using competing risks Cox survival models. We surmised, for example, that a higher rate of injury/accidental death might be indicative of sleep-deprivation attentional deficits. Intriguingly, results revealed that squared age-adjusted sleep was associated with increased likelihood of death from cardiovascular disease (*HR* = 1.20, CIs [1.00, 1.45]) and with death due to infection (*HR* = 1.26, CIs [1.08, 1.47]), for males only, suggesting possible immune system involvement via inflammatory mechanisms (Hotamisligil, 2006; Kemeny, 2011; see Table 6). Childhood sleep duration was not associated with other causes of death (cancer, injury, or other causes), nor was it associated with specific causes of death in the full sample or for females.

Discussion

This study is the first to show links between childhood sleep duration and lifelong mortality risk. We found that healthy male children and adolescents who slept more or less than their peers were at increased risk of dying throughout their adult life span. This is consistent with past studies of sleep, health, and mortality in *adults*, which have found a U-shaped relationship, such that both long and short sleep durations are associated with poorer health and greater mortality risk. However, discovering this relationship across the full life span places previous findings in a new light.

In previous research, sleep duration may have been confounded with undiagnosed chronic comorbid illness. That is, it has been difficult to discern whether sleep is a *marker* of undiagnosed disease—especially in old age—or whether links between sleep and health unfold throughout the entire life span (Grandner & Patel, 2009). For the current sample, the participants were healthy children who lived at least through young adulthood (1940); it is

Table 5
Risk of Death Associated With Different Values of Age and Age-Adjusted Sleep for Male and Female Participants

Age	Age-adjusted sleep (hours)	Risk, males	Risk, females
8	−3	3.13	1.32
8	−2	1.69	1.27
8	−1	1.21	1.25
8	0	1.16	1.27
8	1	1.47	1.34
8	2	2.47	1.46
8	3	5.52	1.64
12	−3	2.71	1.04
12	−2	1.47	.99
12	−1	1.05	.98
12	0	1.00	1.00
12	1	1.27	1.05
12	2	2.13	1.14
12	3	4.77	1.28
16	−3	2.34	.81
16	−2	1.26	.78
16	−1	.91	.77
16	0	.86	.78
16	1	1.10	.83
16	2	1.84	.90
16	3	4.12	1.00

Note. Twelve-year-old male participants sleeping three hours more than their peers were at 4.77 times the mortality risk of those sleeping the average (predicted) amount for their peers.

not likely that child sleep duration was a marker for serious undiagnosed chronic illness in this study.

The current findings suggest that the previously reported associations between sleep and health risk in adulthood are not spurious; rather, even sleep duration in childhood may be an important marker of mortality risk throughout the entire life span. This is in line with the sparse but important findings of previous research, such as Dew et al. (2003), who found that electroencephalographically recorded sleep was a marker of successful aging in an initially healthy older adult sample, and Dahl and Lewin (2002), who found that salubrious sleep is a marker of healthful development and successful adaptation in adolescents. We extend these studies by finding that child and adolescent sleep patterns have health sequelae across the life span.

Cause of death analyses revealed that male children were at increased risk of death due to cardiovascular disease and infection. This finding is consistent with the idea that sleep disturbances may be related to poor physiological regulatory mechanisms involving inflammation, known to be relevant to both cardiovascular disease and immune impairment/infection (Cohen et al., 2009; Grandner & Patel, 2009; Hotamisligil, 2006; Kemeny, 2011). Future research into this potential mediator may prove fruitful.

The difference in effect sizes between the male and female participants in the study is intriguing. Previous research in the Terman data typically yields larger effect sizes for psychosocial variables in men (Friedman & Martin, 2011), often because women's roles and activities were more limited in this cohort (see also Kruger & Nesse, 2006). Previous research on sleep and health in children and adolescents focused on obesity has shown some larger effect sizes for males (e.g., Eisenmann, Ekkekakis, & Holmes, 2006), but other work has found no gender differences (e.g., Landhuis et al., 2008). Sleep duration may be more relevant to male health and mortality but we do not yet know why. Thus, models of sleep and health henceforth might profitably test long-term links between sleep and biopsychosocial wellness that incorporate matters of gender differences in social roles, social networks, health behaviors, and sex hormones.

The Terman sample consists of intelligent, initially healthy, mostly middle-class children, with access to health care and education. This is a limitation on generality but is also simultaneously a research strength, as the lifelong mortality results are likely not due to initial health problems, access to health care, socioeconomic status at baseline, or the ability to understand health recommendations. Compared with large epidemiological studies of sleep and health, the present study has a relatively small sample size (N_{male} = 633; N_{female} = 512), but given the relatively smaller sample size and the very long follow-up time frame (M = 64 years of follow-up), the predictive ability of sleep deviations in the male children is remarkable. It is unknown whether the nonsignificant associations among the females are due to the relatively small sample or are reflective of the more general phenomenon (in adults), sometimes showing greater sleep effects for males.

Data on sleep were collected between 1917 and 1926, at a time when modern sleep actigraphy (movement monitors) and polysomnography (multiparameter physiological recordings) were not available. Thus, sleep was measured primarily via parent reports. Estimates of sleep might actually reflect time in bed, and we could not assess the relative influence of physiological sleep parameters.

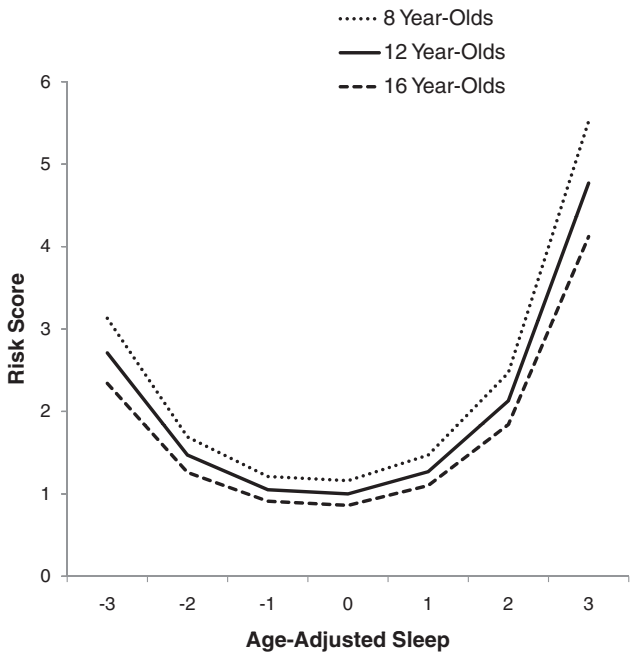

Figure 1. Mortality risk scores for male participants by residualized sleep scores. *Note:* The "0" point in this figure represents individuals who are sleeping the predicted amount (based on regression) as their same-age peers. Individuals with negative values on age-adjusted sleep were sleeping 1, 2, and 3 hr less than their same-age peers, whereas individuals with positive values on age-adjusted sleep were sleeping 1, 2, and 3 hr more than their same-age peers. In this figure, risk scores are stratified based on age at measurement.

Table 6
Cause-Specific Mortality Rates, HR [95% CI]

Cause of death (N)	Centered age	Age-adjusted sleep	Squared age-adjusted sleep
Cardiovascular disease			
Full sample (277)	0.96 [0.92, 1.01]	1 [0.85, 1.18]	1.11 [0.96, 1.27]
Women (117)	0.95 [0.88, 1.02]	0.90 [0.70, 1.16]	1.03 [0.83, 1.28]
Men (160)	0.97 [0.92, 1.02]	1.11 [0.89, 1.37]	1.20 [1.00, 1.45]
Cancer			
Full sample (216)	0.95 [0.91, 1.00]	1.11 [0.92, 1.33]	1.04 [0.88, 1.23]
Women (97)	0.91 [0.84, 0.99]	1.11 [0.86, 1.44]	1.09 [0.87, 1.36]
Men (119)	0.98 [0.92, 1.04]	1.10 [0.85, 1.43]	0.99 [0.77, 1.29]
Injury			
Full sample (51)	0.95 [0.86, 1.05]	1.22 [0.83, 1.77]	1.03 [0.73, 1.45]
Women (19)	1.03 [0.87, 1.22]	1.03 [0.61, 1.75]	1.25 [0.83, 1.87]
Men (32)	0.90 [0.79, 1.02]	1.58 [0.87, 2.86]	0.73 [0.37, 1.42]
Infection			
Full sample (309)	0.95 [0.91, 0.99]	1.13 [0.97, 1.32]	1.08 [0.94, 1.23]
Women (124)	0.97 [0.90, 1.03]	1.30 [0.99, 1.71]	0.84 [0.64, 1.09]
Men (185)	0.94 [0.89, 0.99]	1.10 [0.90, 1.34]	1.26 [1.08, 1.47]
Other			
Full sample (186)	0.97 [0.92, 1.02]	0.98 [0.80, 1.19]	1.07 [0.90, 1.27]
Women (90)	0.92 [0.85, 0.99]	0.91 [0.68, 1.21]	1.02 [0.79, 1.30]
Men (96)	1.02 [0.95, 1.09]	1.05 [0.79, 1.39]	1.14 [0.89, 1.46]

Additionally, because adult sleep duration information is not available, we could not examine associations between childhood sleep, adult sleep, and mortality. However, any error in estimates of sleep duration is theoretically randomly distributed across all participants, and would not affect the direction of our results.

The current study compares sleep duration *within* the sample (rather than using deviance from modern recommendations or average amounts). Physicians and scholars have long been concerned that children are not getting enough sleep (Matricciani, Olds, Blunden, Rigney, & Williams, 2012; Terman & Hocking, 1913). Average sleep duration has indeed been declining (Iglowstein, Jenni, Molinari, & Largo, 2003; Matricciani et al., 2012). Thus, the "average" sleep duration of the Terman participants may not be the "average" sleep duration of modern children. We are not suggesting that it would be salubrious to recommend the "average" amount of sleep in the Terman participants to today's children.

The current findings, demonstrating a six-decade link between childhood sleep duration patterns and mortality risk among males, represent an important extension to previous sleep-mortality research of considerably shorter-term follow-up. This area is ripe for follow-up longitudinal research on mediators. Perhaps most importantly, the results suggest that sleep duration may be in part a core, long-term biobehavioral pattern, thus calling into question models that implicitly assume that associations between sleep deviations and health are primarily indicative of a more transient or shorter-term disturbance.

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