

Loneliness Is Associated with Sleep Fragmentation in a Communal Society

Lianne M. Kurina, PhD¹; Kristen L. Knutson, PhD²; Louise C. Hawkley, PhD³; John T. Cacioppo, PhD³; Diane S. Lauderdale, PhD¹; Carole Ober, PhD⁴

¹Department of Health Studies, ²Department of Medicine, ³Department of Psychology and Center for Cognitive and Social Neuroscience, and ⁴Department of Human Genetics, University of Chicago, Chicago, IL

Study Objective: Loneliness has been shown to predict poor health. One hypothesized mechanism is that lonely individuals do not sleep as well as individuals who feel more connected to others. Our goal was to test whether loneliness is associated with sleep fragmentation or sleep duration.

Design: Cross-sectional study.

Setting: Members of a traditional, communal, agrarian society living in South Dakota.

Participants: Ninety-five participants (mean age 39.8 years, 55% female) who were ≥ 19 years of age at the study's inception.

Interventions: Not applicable.

Measurements and Results: We conducted interviews querying loneliness, depression, anxiety, and stress, as well as subjective sleep quality and daytime sleepiness. Study participants wore a wrist actigraph for one week to measure objective sleep properties; the two studied here were sleep fragmentation and sleep duration. Higher loneliness scores were associated with significantly higher levels of sleep fragmentation ($\beta = 0.073$, $t = 2.55$, $P = 0.01$), controlling for age, sex, body mass index, risk of sleep apnea, and negative affect (a factor comprising symptoms of depression and anxiety, and perceived stress). Loneliness was not associated with sleep duration or with either subjective sleep measure.

Conclusions: Loneliness was a significant predictor of sleep fragmentation. Humans' social nature may partly be manifest through our dependence on feeling secure in our social environment to sleep well.

Keywords: Loneliness, sleep, actigraphy

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INTRODUCTION

Human beings are a social species. The increased risks of morbidity and mortality conferred by deficits in social connections—either objective or perceived—illustrate the importance of our social nature to our health and well-being.¹⁻⁶ Perceived isolation, or loneliness, has been associated with increased blood pressure,⁷ increased risk of heart disease in women,^{8,9} and increased risks of depression,³ cognitive decline,^{2,4} and mortality.¹⁰⁻¹⁴ What are the pathways through which loneliness might affect health? One hypothesis is that sleep, a key restorative behavior, could be compromised by feelings of loneliness.¹⁵ Compromised sleep—including short duration and poor quality sleep—has been linked with various health outcomes, including poorer metabolic function, increased cardiovascular risk, poorer self-rated health, and cognitive and behavioral impairments.¹⁶⁻²³

The formation of meaningful social connections is an integral part of the human experience.^{24,25} Loneliness is typically defined as the painful experience that accompanies a discrepancy between a person's desired and actual social relationships.²⁵ Although sometimes considered synonymous with social isolation, loneliness and social isolation are distinct concepts. The latter reflects an objective measure of social interactions and relationships, whereas loneliness reflects perceived social isolation or feelings of being an outcast. Accordingly, loneliness is more closely associated with the quality rather than the number of relationships.^{26,27}

We posit that the experience of loneliness is particularly salient to sleep because of humans' presumed need to feel safe in order to sleep soundly. Cross-sectional studies have shown that individuals who report greater loneliness also report poorer sleep quality.^{15,28,29} The results of longitudinal studies suggest that loneliness predicts decrements in subjective sleep quality, but not vice-versa.^{18,30} Subjective measures of sleep, however, may be tapping different dimensions of sleep compared to objective measures. Self-reported sleep duration is typically overreported relative to objective measures³¹ and self-reported sleep quality may be influenced by non-sleep factors. For example, women typically have more sleep complaints than men³² and yet get more sleep per night and have better sleep efficiency according to objective measures.³³ Individuals with paradoxical insomnia may report poor quality sleep and yet have normal sleep according to polysomnography.³⁴ It is therefore useful to collect both objective and subjective measures, with the objective measures having the advantage of more straightforward interpretation.

The one study that tested for associations between social factors and objective sleep properties, conducted in a convenience sample of college students, showed that loneliness is associated with poorer objective sleep efficiency but not differences in sleep duration.³⁵ That is, loneliness does not appear to change the total *amount* of sleep an individual accrues, but rather, it is associated with more microawakenings during the night.³⁵

From an evolutionary perspective, this finding makes sense; humans must have relied on a safe social surround to survive and thrive. The absence of a secure social environment results in perceptions of social isolation that, in turn, have been shown to increase vigilance for threat and to heighten feelings of vulnerability.³⁶ This heightened vigilance may be manifested during the sleep period as greater restlessness or more fragmented sleep.

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Address correspondence to: Lianne M. Kurina, 5841 S. Maryland Ave., MC 2007, Chicago IL 60637; Tel: (773) 834-3926; Fax: (773) 702-1979; E-mail: lkurina@uchicago.edu

Table 1—Description of the study population (N = 95)

Characteristic	Mean (SD)	Range
Age, years	39.8 (14.2)	19–84
Sex (% female)	55	—
Body mass index (kg/m ²)	27.8 (4.8)	17.5–41.3
Obesity (%)	26	—
Hypertension (%)	30	—
Risk of sleep apnea (%)	14	—
Loneliness ^a	0.69 (1.1)	0–5
Depressive symptoms ^b	4.6 (2.6)	0–14
Symptoms of anxiety ^c	5.0 (3.5)	0–13
Perceived stress ^d	5.3 (3.2)	0–15
Sleep fragmentation ^e (%)	31.3 (9.2)	14.2–63.2
Sleep duration (hours, actigraphy-derived)	6.3 (0.6)	4.7–8.6
Subjective sleep quality ^f	5.7 (3.5)	0–20
Subjective daytime sleepiness ^g	5.5 (3.0)	0–13
Average reported weekday bedtime (PSQI)	22.6 (0.8)	21–26
Average reported weekday waketime (PSQI)	6.6 (0.6)	3.8–9.0

^aMeasure derived from the R-UCLA Loneliness Scale;^{43,44} scores can range from 0–6. ^bBased on the MHI-5,⁴⁵ scores can range from 0–20. ^cBased on the anxiety-related items from the HADS scale;⁴⁶ scores can range from 0–21. ^dBased on 4 items from the PSS;⁴⁸ scores can range from 0–16. ^eSleep fragmentation is the sum of the percentage of the sleep period spent moving and the percentage of the number of immobile phases that are only one-minute long. ^fSubjective sleep quality is measured with the PSQI;⁵⁷ scores can range from 0–21, and higher scores indicate poorer sleep quality. ^gSubjective daytime sleepiness is measured with a modified version of the ESS;⁵⁹ scores can range from 0–18, and higher scores indicate greater sleepiness.

Given the limited literature on loneliness in relation to objective sleep properties, the primary goal of this study was to test the hypothesis that variation in feelings of loneliness would be associated with variation in sleep fragmentation—in a very different study population than that previously investigated. We also tested for associations between loneliness and total sleep duration and between loneliness and self-reported sleep quality and daytime sleepiness.

Specifically, we studied sleep and loneliness in a group of Hutterites. The study participants are part of a traditional, agrarian, communal society, a group we might expect to be among the most socially connected in the United States. Further, because of the remarkable lifestyle uniformity in the Hutterites, the results are protected against the confounding influences of SES and tobacco that can be problematic for tests of these hypotheses conducted in the general population. Other psychological phenotypes such as depression, anxiety, and stress, are known to cluster with loneliness and may themselves have effects on sleep.^{3,30,37} We therefore measured these phenotypes in a structured interview and controlled for their effects in the analyses.

METHODS

Study Design and Subjects

Our study of stress and sleep took place within the context of an established genetic-epidemiologic cohort study of the Hutterites that has been ongoing for over two decades.^{38–41} The

Hutterites are an Anabaptist group of European descent that originated in the 16th century. Three Hutterite communal farms (called “colonies”) were established in the Dakota territories in the late 1800s, and the current Hutterite population in North America numbers more than 30,000.⁴² Communal living is central to the Hutterites’ religious philosophy, and they follow a traditional lifestyle with clearly defined and stratified gender roles.⁴² Hutterite colonies consist of approximately 150 individuals and are large agricultural enterprises. Hutterites share all resources in common, follow a similar schedule, work together, and share the same diet. Tobacco use is prohibited. Therefore, this group offers near uniformity in a number of key socioeconomic and lifestyle variables.

The study population for the stress and sleep study consisted of eligible Hutterite adults—that is, those 19 years of age and older—living on two colonies in South Dakota. During the winter of 2006–7, we collected data on psychosocial factors and sleep as well as anthropometric and blood pressure measurements from study participants. Data on loneliness, depression, anxiety, stress, subjective sleep quality, and subjective daytime sleepiness were collected via structured interviews from 130 individuals. Additionally, alcohol consumption questions were included in the interview for one of the two colonies. One hundred fifty-three adults from the two farms were eligible to participate, and the initial participation rate was therefore 85%. In order to measure objective sleep properties, the study employed wrist actigraphy; the procedures followed are described in more detail below. Ninety-seven actigraphy devices were available, and these were offered consecutively to individuals following the interview. Eleven

individuals of those initially offered declined, resulting in an actigraphy uptake rate of 90%. Although 97 individuals wore a wrist actigraph for one week, one individual’s anthropometric data were deemed unreliable, and blood pressure data were unavailable for a second. Our analytic sample therefore includes 95 individuals with complete interview, actigraphy, anthropometric, and blood pressure data (Table 1). All participants provided written informed consent and the study protocol was approved by the Institutional Review Board of the University of Chicago.

Psychosocial Measures

Loneliness

Loneliness was measured with the 3-item loneliness scale derived from the R-UCLA Loneliness Scale.^{43,44} Specifically, participants were asked how often they felt they lacked companionship, felt left out, and felt isolated from others. Responses were on a scale from 0 (hardly ever) to 2 (often). Cronbach α for this measure was 0.69 in this sample, similar to the value (0.72) reported for the larger population-based sample in which the measure was developed.⁴⁴

Depressive symptoms

Depressive symptoms were measured using the Mental Health Index 5 (MHI-5) scale,⁴⁵ which queries feelings experienced during the past month. Responses to the 5 items were

on a scale from 0 (none of the time) to 4 (all of the time). Cronbach α for this measure was 0.78.

Symptoms of anxiety

Symptoms of anxiety were measured using the 7 anxiety items from the Hospital Anxiety and Depression Scale (HADS),^{46,47} which queries feelings experienced during the past week. Responses were on a scale from 0 (none of the time) to 3 (most of the time). Cronbach α for the measure was 0.76.

Perceived stress

Perceived stress was originally measured using 6 items from Cohen's perceived stress scale (PSS),⁴⁸ which queries feelings experienced during the past month. Because interviewers perceived confusion among respondents for one of the items and because an additional item was discordant with cultural precepts, the measure of perceived stress used in the analyses comprised 4 items, 2 of which overlap with the more typically used 4-item PSS.⁴⁸ Responses were on a scale from 0 (never) to 4 (very often). Cronbach α for this measure was 0.81.

Negative affect

We expected that depressive symptoms, symptoms of anxiety, and perceived stress would be highly correlated and this expectation was born out (Table 2). For simplicity's sake, we used factor analysis to condense these 3 variables. The principal-factor method yielded a single factor, which we will refer to as "negative affect," with an eigenvalue of 1.65. Depressive symptoms, symptoms of anxiety, and perceived stress had loading values of 0.73, 0.73, and 0.76, respectively.

Alcohol Consumption

In one of the study colonies, 3 questions were included in the interview to garner information on typical alcohol consumption. Study participants were asked whether they ever drank alcoholic beverages, the number of days per week over the last 3 months that they typically consumed alcohol, and the number of drinks per day they consumed on the days that they drank alcohol. These questions were used to calculate a typical number of drinks per week consumed by each study participant. Because alcohol consumption data were only collected for half of the sample, these data were used in exploratory analyses, but not in the final models.

Objective Sleep Measures

Sleep duration and fragmentation were measured with wrist actigraphy (Actiwatch model AW-16, Philips/Respironics, Bend, OR) using 1-minute epochs. Actiwatches are small, watch-like devices that have sensitive omnidirectional accelerometers that digitally record an integrated measure of gross motor activity, which is analyzed to identify sleep periods. Participants were instructed to wear the Actiwatch continuously for 7 days. The Actiwatch has an event marker button that participants were instructed to press to record bedtimes and wake times. Actiwatches have been validated against polysomnography; for healthy adults, reported correlations with total sleep time as assessed by polysomnography are as high as 0.97.⁴⁹⁻⁵¹

Table 2—Correlations among loneliness, depressive symptoms, symptoms of anxiety, perceived stress, and negative affect (N = 95)

	Loneliness	Depressive symptoms	Symptoms of anxiety	Perceived stress
Depressive symptoms	0.30**			
Symptoms of anxiety	0.32**	0.60***		
Perceived stress	0.14	0.69***	0.64***	
Negative effect	0.28**	0.86***	0.85***	0.90***

P < 0.01; *P < 0.001.

Actigraphy yields a number of different metrics for sleep, some of which require an accurate indicator of when the subject began to try to fall asleep, which we were missing from some subjects on some nights. For our outcome measures, we chose not use the metrics that depended strongly on that information and focused on 2 metrics—sleep duration and sleep fragmentation—that are moderately correlated ($r = -0.37$, $P < 0.001$). We limited the analyses to these 2 measures because our primary hypothesis concerned fragmentation and we wished to avoid redundancy and control the number of statistical tests conducted. These 2 sleep measures were derived from the Actiwatch recording using the manufacturer's software algorithms (Actiware 3.4). An average value over all of the days of actigraphy recordings was calculated for each sleep measure for each individual.

Sleep fragmentation

Fragmentation is an index of restlessness during the sleep period expressed as a percentage. It is defined by the manufacturer's software as the sum of 2 percentages: (1) the percentage of the sleep period spent moving (an epoch with more than 2 activity counts is considered moving), and (2) the percentage of the number of immobile phases (i.e., an epoch with no movement) that are only one minute long.

Fragmentation is an appealing actigraphy metric because it is an indicator of sleep disruption or disturbance^{52,53} and because it most closely approximates the polysomnography measure of microarousals, which have been shown to be associated with poor health outcomes.⁵⁴ Further, actigraphy-defined sleep fragmentation has now been used in several studies of sleep and health.^{16,55,56}

Sleep duration

This is the total amount of sleep obtained at night (expressed in hours). It excludes periods of wakefulness between bedtime and wake time.

Subjective Sleep Quality Measures

Pittsburgh Sleep Quality Index (PSQI)

The PSQI is designed to assess sleep quality during the past month, and contains 19 self-rated questions from which 7 component scores are calculated and summed into a global score.⁵⁷ Higher scores represent worse sleep quality: component scores range from 0 to 3, and global scores range from 0 to 21. Previous studies have demonstrated good test-retest reliability for

the PSQI, with correlation coefficients of 0.85 and 0.87 over a period of 2 days to a month.^{57,58} The PSQI also includes questions about respondents' usual bedtime and usual arising time for both weekdays and weekend days.

Epworth Sleepiness Scale (ESS)

The ESS is an 8-item questionnaire designed to assess an individual's level of daytime sleepiness, and scores on this instrument range from 0-24, where higher scores indicate greater sleepiness.⁵⁹ A test-retest reliability study of the ESS yielded a Pearson correlation of 0.82 over a 5-month interval.⁶⁰ In this study, we employed a modified ESS from which 2 questions were removed because they were not relevant to the study population's experience, i.e., how likely the respondent was to fall asleep while watching TV or while in a car, while stopped for a few minutes in traffic. There are no TVs on the Hutterite colonies, and car travel is rare, especially for women. We then used the modified scale to identify excessive daytime sleepiness. Excessive daytime sleepiness is typically defined as a score ≥ 10 of a possible range of 24 on the standard ESS.⁵⁹ On our modified scale, therefore, individuals scoring ≥ 8 (of a possible 18) were classified as having excessive daytime sleepiness; 23% of the study population had scores ≥ 8 .

Body Mass Index

Body mass index (BMI, kg/m²) was calculated for each of the study participants and included in the analyses because of its cross-sectional associations with the sleep outcomes of interest.⁵⁶ Height was measured to the nearest 3 mm with a plastic stadiometer with the participants in stocking feet. Weight was measured with a Health-O-Meter 349KL digital medical scale with the subject wearing light clothing. BMI was modeled as a continuous variable in the analyses.

Blood Pressure

Nurses used mercury-gravity sphygmomanometers to measure blood pressure for each individual. Blood pressure measurements were taken on each individual in a seated position 2 times, with a 2-minute interval between the 2 measurements, following an initial 5-minute rest period.⁶¹

Study participants were asked to bring all regularly used medications (both prescription and non-prescription) with them. Medication names together with indication, dosage, and frequency of use were recorded for all study participants.

Sleep Apnea Risk

Controlling for sleep apnea risk is important because of its possible associations with the sleep outcomes.⁶² Individuals were classified as at risk of having sleep apnea if they had ≥ 2 of the 3 following conditions: snoring; excessive daytime sleepiness; and obesity or hypertension. Individuals were considered to snore if they endorsed having trouble sleeping ≥ 3 times per week during the last month because they either "could not breathe comfortably" or "coughed or snored loudly" on the PSQI.⁵⁷ Excessive daytime sleepiness was identified using the modified ESS⁵⁹ as described above. Per convention, individuals with a BMI ≥ 30 kg/m² were classified as obese. Finally, study participants were classified as hypertensive if they had elevated blood pressure at the physical exam (≥ 130

mm Hg systolic or ≥ 85 mm Hg diastolic) or were on drug treatment for hypertension.

Analytic Strategy

The primary outcome of interest in the study was sleep fragmentation and the secondary outcomes were sleep duration, subjective sleep quality, and subjective daytime sleepiness. All outcomes were treated as continuous variables. The fragmentation data were normalized with a natural log transform and the PSQI data were normalized with a square root transform. Because the Hutterites are a founder population, with some relatedness among individuals, we used mixed linear models (SAS PROC MIXED) to test for associations between the sleep outcomes and loneliness. In order to take into account the relatedness between individuals, a random genetic effect was modeled with mean zero and a covariance structure that is dependent on the relationships between study participants, using a kinship coefficient developed from the population's pedigree.^{63,64}

Two models were run for each of the sleep outcomes. The first model was a simple regression with loneliness, modeled continuously, as the sole predictor of the sleep outcome, controlling for relatedness among individuals. The second set of models included age, sex, BMI, risk of sleep apnea, and negative affect. Risk of sleep apnea was omitted as a predictor of daytime sleepiness, because of the strong collinearity between the two. Age, BMI, and negative affect were modeled continuously, and sex and risk of sleep apnea were included as dichotomous variables. Sensitivity analyses were run to test for effects of including objective social connections and alcohol consumption as predictors on the associations of interest between loneliness and the sleep outcomes. We also tested for effects of compliance with use of actigraphy event markers. Descriptive statistics and correlations were calculated using Stata version 11.1 (StataCorp, College Station, TX). The mixed linear models were run with SAS version 9.1 (SAS, Cary, NC).

RESULTS

Table 1 presents a descriptive summary of the demographic, physical, psychological, and sleep-related characteristics of the study population. There were approximately equal numbers of men and women, and age distributions in the two sexes were similar. Half of the study population was between 19 and 36 years of age, the third quartile was between 37 and 47 years of age, and the top quartile was between 48 and 84 years of age.

Overall, on average, we observed low levels of loneliness and low levels of depressive or anxious symptomatology (Table 1). Bedtimes and arising times were generally uniform; for example, 90% of the sample reported going to bed on weekdays between 21:30 and 23:30, and all but two individuals reported arising on weekdays between 05:00 and 07:00. Average bedtimes and arising times for weekends were similar to those for weekdays.

Table 2 presents the correlations among the psychosocial variables. As noted earlier, depressive symptoms, symptoms of anxiety, and perceived stress were all strongly and significantly related to each other, with correlation coefficients between 0.6 and 0.7. As expected, each of these psychosocial variables was also strongly related to the negative affect factor derived from them. Loneliness shows low to moderate correlations with de-

pression, anxiety, stress, and negative affect; this is evidence that despite its ties to these variables, loneliness is also capturing a distinct aspect of humans' psychological experience.

Table 3 presents the results of the analyses regressing the 4 sleep outcomes on loneliness. In these simple models, loneliness was significantly associated with both objective sleep measures but was not associated with either subjective sleep measure.

Table 4 presents results of the multivariate models including loneliness and all control variables as predictors. Each unit increase in the UCLA loneliness scale was associated with an approximately 8% increase in sleep fragmentation ($\beta = 0.073$, $t = 2.55$, $P = 0.01$). Loneliness was not significantly associated with sleep duration, self-reported sleep quality, or self-reported daytime sleepiness in these models (Table 4). Negative affect was significantly associated with subjective sleep quality, but not with any of the other sleep outcomes. Females had significantly lower levels of sleep fragmentation and reported daytime sleepiness than did males. Finally, age was positively and significantly associated with sleep duration. There was, however, one influential observation, and when this individual was removed from the analysis, the association between age and sleep duration was no longer significant.

Adding sleep duration, objective measures of social connectedness (i.e., marital status or the number of family members living in the same colony), and alcohol consumption (for the subset of study participants with consumption data) as predictors to the sleep fragmentation model had no material effect on the association between loneliness and sleep fragmentation. Restricting the analysis to those individuals who used their actigraphy event markers $\geq 85\%$ of the time also resulted in no change to our loneliness/sleep outcomes associations.

DISCUSSION

In this study, lonely individuals experienced significantly more sleep fragmentation than did those who reported more connection to others, suggesting that perceptions of a secure social surround may promote a better, more restful night's sleep. On average, each unit increase in loneliness was associated with an 8% increase in sleep fragmentation. In contrast, we did not observe an association between loneliness and sleep duration. Our findings replicate those of an earlier study³⁵—conducted in a very different sample—and lend support to the hypothesis that sleep quality, at least as indexed by sleep fragmentation,

may be one pathway through which feelings of social connectedness influence future health.

The first study of perceived social connectedness and objective sleep properties utilized a sample of college students,³⁵ a younger and more ethnically and socioeconomically diverse population occupying a very different social space compared with the Hutterites. Social networks are dynamic in college and the undergraduates in the study were selected to represent the spectrum of loneliness. In contrast, Hutterites follow a communal lifestyle notable for frequent interaction (e.g., working and eating together). Their lives revolve around a community with strong residential stability and presumably deep social ties of long duration. Finally, among the present study population, approximately half of the group was between 19 and 40 years of age and the other half between 40 and 84 years of age.

Even given the pronounced differences in the study populations, effectively identical results were obtained in the two studies. Individuals who reported greater feelings of loneliness had more fragmented sleep, but the same amount of total sleep, compared with those who reported greater perceived social connectedness. This effect persisted in the present study even when controlling for negative affect, pointing to a particularly salient link between feelings of loneliness and sleeping soundly.

Our findings are particularly noteworthy because levels of loneliness in this population are quite low compared to those in other populations, including a nationally representative sample of individuals 50 years of age and older⁴⁴ as well as that of a college-aged sample (L. Hawkey, personal communication, June 4, 2010). What our findings suggest, therefore, is that the relationship between loneliness and sleep is consistent across the range of perceived connectedness and is not just the product

Table 3—Results of simple models regressing the 4 sleep outcomes on loneliness (N = 95)

Sleep Outcome	β (SE)
Sleep fragmentation (ln-transformed)	0.064 (0.028)*
Sleep duration	-0.13 (0.06)*
Subjective sleep quality (square root-transformed)	0.095 (0.071)
Subjective daytime sleepiness	0.36 (0.27)

* $P < 0.05$.

Table 4—Results of mixed linear models analyzing loneliness in relation to the 4 sleep outcomes controlling for demographic and health-related factors as well as negative affect (N = 95)

Predictor	Sleep fragmentation (ln-transformed)	Sleep duration	Subjective sleep quality (sqrt-transformed)	Subjective daytime sleepiness
	β (SE)	β (SE)	β (SE)	β (SE)
Loneliness	0.073 (0.029)*	-0.089 (0.064)	0.020 (0.068)	0.45 (0.26)
Age (years)	0.00052 (0.0022)	0.011 (0.0052)*	0.0040 (0.0057)	0.0054 (0.02)
Sex (reference = males)	-0.22 (0.066)**	-0.045 (0.14)	0.069 (0.15)	-2.84 (0.59)***
BMI (kg/m ²)	0.0030 (0.0072)	-0.015 (0.016)	-0.0096 (0.017)	-0.018 (0.06)
Risk of sleep apnea	-0.107 (0.091)	-0.062 (0.20)	-0.33 (0.22)	-
Negative affect	0.038 (0.034)	-0.059 (0.076)	0.35 (0.08)***	0.55 (0.30)

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

of comparing very lonely with very connected individuals. The replication of the association between loneliness and sleep fragmentation across the two disparate samples indicate that this finding may be more widely generalizable.

It is possible, of course, that it is not loneliness that increases sleep fragmentation, but that individuals with more fragmented sleep then suffer from greater feelings of disconnection. Roberts et al., for example, documented prospective effects of insomnia on interpersonal functioning among adolescents.⁶⁵ We cannot infer directionality in this study, given the cross-sectional nature of the data. However, it may be telling that sleep fragmentation is most strongly associated with feelings of social connection versus, for example, negative affect. If the effect were mainly from sleep to psychological symptoms, it is not obvious why the effect would be stronger for perceived connection than for negative affect. We would also draw attention to a longitudinal study of loneliness and subjective sleep quality conducted across three days which showed that while loneliness decreased future subjective sleep quality, subjective sleep quality did not influence loneliness.³⁰

Interestingly, the differences in sleep observed are not due to differences in health behaviors (i.e., getting more sleep). Rather, it is the soundness of sleep that appears to be compromised by feelings of loneliness. Adding sleep duration as a predictor to the sleep fragmentation model had no effect on the association between loneliness and sleep fragmentation. It is also worth noting that it is the *perception* of connectedness that is driving the association. When we added more objective measures of social connectedness (i.e., marital status or the number of family members living in the same colony) as control variables to the analysis, the association between loneliness and sleep fragmentation was unchanged.

Known effects of alcohol and caffeine on sleep^{66,67} prompted us to consider these in our study. Our exploratory analyses showed no change in the association between loneliness and the sleep outcomes when alcohol consumption was added to the model for the subset of participants for whom the data were available. Hutterite adults do typically drink a lot of coffee but, unfortunately, we did not formally assess caffeine consumption in our study.

One concern using actigraphy data is that sleep duration may be overestimated when individuals lie very quietly, but are awake; this may be particularly challenging when event markers are not used. Sensitivity analyses showed that our results were unchanged even when restricted to the subset of participants who used event markers the majority ($\geq 85\%$) of the time. If lonely people were more likely to lie very still (without sleeping), their sleep duration could be overestimated and sleep fragmentation underestimated. However, very high correlations—up to 0.97—have been reported between actigraphy and polysomnography for total sleep time.^{49,50}

In contrast to some past studies,^{15,30} neither subjective sleep quality nor subjective daytime sleepiness was associated with loneliness in either the simple or adjusted models. This may be because we had insufficient range in subjective sleep quality in this population. While we found that both the global PSQI score and the daytime dysfunction component score of the PSQI were higher in lonely compared to non-lonely individuals in our

study, consistent with reports in past studies,¹⁵ the Hutterites had lower scores on these measures overall.

This study was partly motivated by the substantial literature documenting ill health effects of loneliness. Could greater sleep fragmentation among lonelier individuals help to explain this association? Cross-sectional associations between sleep fragmentation and BMI^{56,68} and type 2 diabetes⁶⁹ have been reported. Past experimental studies conducted in humans and animals have documented hormonal and behavioral effects of induced sleep fragmentation.⁷⁰ Recent experimental studies in humans have shown that fragmented sleep had a negative impact on glucose metabolism and caused increased sympathetic nervous system activity.^{71,72} It is plausible, therefore, that sleep fragmentation may be a link between loneliness and downstream health outcomes.

In summary, our study provides evidence that those individuals who perceive themselves as less connected to others have more fragmented sleep. Sleep could be a pathway through which perceived social isolation influences health.

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DISCLOSURE STATEMENT

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REFERENCES

1. House JS, Landis KR, Umberson D. Social relationships and health. *Science* 1988;241:540-5.
2. Tilvis RS, Kahonen-Vare MH, Jolkkonen J, Valvanne J, Pitkala KH, Strandberg TE. Predictors of cognitive decline and mortality of aged people over a 10-year period. *J Gerontol A Biol Sci Med Sci* 2004;59:268-74.
3. Cacioppo JT, Hughes ME, Waite LJ, Hawkley LC, Thisted RA. Loneliness as a specific risk factor for depressive symptoms: cross-sectional and longitudinal analyses. *Psychol Aging* 2006;21:140-51.
4. Wilson RS, Krueger KR, Arnold SE, et al. Loneliness and risk of Alzheimer disease. *Arch Gen Psychiatry* 2007;64:234-40.
5. Holt-Lunstad J, Smith TB, Layton JB. Social relationships and mortality risk: a meta-analytic review. *PLoS Med* 2010;7:e1000316.
6. Seeman TE. Health promoting effects of friends and family on health outcomes in older adults. *Am J Health Promot* 2000;14:362-70.
7. Hawkley LC, Thisted RA, Masi CM, Cacioppo JT. Loneliness predicts increased blood pressure: 5-year cross-lagged analyses in middle-aged and older adults. *Psychol Aging* 2010;25:132-41.
8. Eaker ED, Pinsky J, Castelli WP. Myocardial infarction and coronary death among women: psychosocial predictors from a 20-year follow-up of women in the Framingham Study. *Am J Epidemiol* 1992;135:854-64.
9. Thurston RC, Kubzansky LD. Women, loneliness, and incident coronary heart disease. *Psychosom Med* 2009;71:836-42.
10. Patterson AC, Veenstra G. Loneliness and risk of mortality: A longitudinal investigation in Alameda County, California. *Soc Sci Med* 2010.
11. Penninx BW, van Tilburg T, Kriegsman DM, Deeg DJ, Boeke AJ, van Eijk JT. Effects of social support and personal coping resources on mortality in older age: the Longitudinal Aging Study Amsterdam. *Am J Epidemiol* 1997;146:510-9.
12. Sugisawa H, Liang J, Liu X. Social networks, social support, and mortality among older people in Japan. *J Gerontol* 1994;49:S3-13.
13. Shiovitz-Ezra S, Ayalon L. Situational versus chronic loneliness as risk factors for all-cause mortality. *Int Psychogeriatr* 2010;22:455-62.

14. Olsen RB, Olsen J, Gunner-Svensson F, Waldstrom B. Social networks and longevity. A 14 year follow-up study among elderly in Denmark. *Soc Sci Med* 1991;33:1189-95.
15. Cacioppo JT, Hawkley LC, Crawford LE, et al. Loneliness and health: potential mechanisms. *Psychosom Med* 2002;64:407-17.
16. King CR, Knutson KL, Rathouz PJ, Sidney S, Liu K, Lauderdale DS. Short sleep duration and incident coronary artery calcification. *JAMA* 2008;300:2859-66.
17. Gangwisch JE, Heymsfield SB, Boden-Albala B, et al. Short sleep duration as a risk factor for hypertension: analyses of the first National Health and Nutrition Examination Survey. *Hypertension* 2006;47:833-9.
18. Jacobs JM, Cohen A, Hammerman-Rozenberg R, Stessman J. Global sleep satisfaction of older people: the Jerusalem Cohort Study. *J Am Geriatr Soc* 2006;54:325-9.
19. Ohayon MM. Prevalence and correlates of nonrestorative sleep complaints. *Arch Intern Med* 2005;165:35-41.
20. Roth T, Jaeger S, Jin R, Kalsekar A, Stang PE, Kessler RC. Sleep problems, comorbid mental disorders, and role functioning in the national comorbidity survey replication. *Biol Psychiatry* 2006;60:1364-71.
21. Jennings JR, Muldoon MF, Hall M, Buysse DJ, Manuck SB. Self-reported sleep quality is associated with the metabolic syndrome. *Sleep* 2007;30:219-23.
22. Nilsson PM, Roost M, Engstrom G, Hedblad B, Berglund G. Incidence of diabetes in middle-aged men is related to sleep disturbances. *Diabetes Care* 2004;27:2464-9.
23. Kawakami N, Takatsuka N, Shimizu H. Sleep disturbance and onset of type 2 diabetes. *Diabetes Care* 2004;27:282-3.
24. Baumeister RF, Leary MR. The need to belong: desire for interpersonal attachments as a fundamental human motivation. *Psychol Bull* 1995;117:497-529.
25. Cacioppo JT, Patrick W. Loneliness: human nature and the need for social connection. New York: WW Norton, 2008.
26. Peplau LA, Perlman D. Loneliness: a sourcebook of current theory, research, and therapy. New York: Wiley, 1982.
27. Wheeler L, Reis H, Nezlek J. Loneliness, social interaction, and sex roles. *J Pers Soc Psychol* 1983;45:943-53.
28. Allaert FA, Urbinelli R. Sociodemographic profile of insomniac patients across national surveys. *CNS Drugs* 2004;18 Suppl 1:3-7;discussion 41, 3-5.
29. Mahon NE. Loneliness and sleep during adolescence. *Percept Mot Skills* 1994;78:227-31.
30. Hawkley LC, Preacher KJ, Cacioppo JT. Loneliness impairs daytime functioning but not sleep duration. *Health Psychol* 2010;29:124-9.
31. Lauderdale DS, Knutson KL, Yan LL, Liu K, Rathouz PJ. Self-reported and measured sleep duration: how similar are they? *Epidemiology* 2008;19:838-45.
32. Groeger JA, Zijlstra FR, Dijk DJ. Sleep quantity, sleep difficulties and their perceived consequences in a representative sample of some 2000 British adults. *J Sleep Res* 2004;13:359-71.
33. Lauderdale DS, Knutson KL, Yan LL, et al. Objectively measured sleep characteristics among early-middle-aged adults: the CARDIA study. *Am J Epidemiol* 2006;164:5-16.
34. Parrino L, Milioli G, De Paolis F, Grassi A, Terzano MG. Paradoxical insomnia: the role of CAP and arousals in sleep misperception. *Sleep Med* 2009;10:1139-45.
35. Cacioppo JT, Hawkley LC, Bernston GG, et al. Do lonely days invade the nights? Potential social modulation of sleep efficiency. *Psychol Sci* 2002;13:384-7.
36. Cacioppo JT, Hawkley LC. Perceived social isolation and cognition. *Trends Cogn Sci* 2009;13:447-54.
37. Mezick EJ, Matthews KA, Hall M, et al. Intra-individual variability in sleep duration and fragmentation: associations with stress. *Psychoneuroendocrinology* 2009;34:1346-54.
38. Gallego Romero I, Ober C. CFTR mutations and reproductive outcomes in a population isolate. *Hum Genet* 2008;122:583-8.
39. Ober C, Abney M, McPeck MS. The genetic dissection of complex traits in a founder population. *Am J Hum Genet* 2001;69:1068-79.
40. Ober C, Tan Z, Sun Y, et al. Effect of variation in CHI3L1 on serum YKL-40 level, risk of asthma, and lung function. *N Engl J Med* 2008;358:1682-91.
41. Weiss LA, Pan L, Abney M, Ober C. The sex-specific genetic architecture of quantitative traits in humans. *Nat Genet* 2006.
42. Hostetler JA. Hutterite Society. Baltimore: The Johns Hopkins University Press, 1974.
43. Russell D, Peplau LA, Cutrona CE. The revised UCLA Loneliness Scale: concurrent and discriminant validity evidence. *J Pers Soc Psychol* 1980;39:472-80.
44. Hughes ME, Waite LJ, Hawkley LC, Cacioppo JT. A short scale for measuring loneliness in large surveys: Results from two population-based studies. *Res Aging* 2004;26:665-72.
45. Berwick DM, Murphy JM, Goldman PA, Ware JE Jr, Barsky AJ, Weinstein MC. Performance of a five-item mental health screening test. *Med Care* 1991;29:169-76.
46. Herrmann C. International experiences with the Hospital Anxiety and Depression Scale--a review of validation data and clinical results. *J Psychosom Res* 1997;42:17-41.
47. Bjelland I, Dahl AA, Haug TT, Neckelmann D. The validity of the Hospital Anxiety and Depression Scale. An updated literature review. *J Psychosom Res* 2002;52:69-77.
48. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. *Journal of Health and Social Behavior* 1983;24:385-96.
49. Ancoli-Israel S, Cole R, Alessi C, Chambers M, Moorcroft W, Pollak CP. The role of actigraphy in the study of sleep and circadian rhythms. *Sleep* 2003;26:342-92.
50. Jean-Louis G, von Gizycki H, Zizi F, et al. Determination of sleep and wakefulness with the actigraph data analysis software (ADAS). *Sleep* 1996;19:739-43.
51. Jean-Louis G, von Gizycki H, Zizi F, Spielman A, Hauri P, Taub H. The actigraph data analysis software: I. A novel approach to scoring and interpreting sleep-wake activity. *Percept Mot Skills* 1997;85:207-16.
52. Loewen A, Siemens A, Hanly P. Sleep disruption in patients with sleep apnea and end-stage renal disease. *J Clin Sleep Med* 2009;5:324-9.
53. Rimmer J, Downie S, Bartlett DJ, Gralton J, Salome C. Sleep disturbance in persistent allergic rhinitis measured using actigraphy. *Ann Allergy Asthma Immunol* 2009;103:190-4.
54. Thomas RJ. Sleep fragmentation and arousals from sleep-time scales, associations, and implications. *Clin Neurophysiol* 2006;117:707-11.
55. Knutson KL, Van Cauter E, Zee P, Liu K, Lauderdale DS. Cross-sectional associations between measures of sleep and markers of glucose metabolism among subjects with and without diabetes: The Coronary Artery Risk Development in Young Adults (CARDIA) Sleep Study. *Diabetes Care* 2011;34:1171-6.
56. Lauderdale DS, Knutson KL, Rathouz PJ, Yan LL, Hulley SB, Liu K. Cross-sectional and longitudinal associations between objectively measured sleep duration and body mass index: the CARDIA Sleep Study. *Am J Epidemiol* 2009;170:805-13.
57. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res* 1989;28:193-213.
58. Backhaus J, Junghanns K, Broocks A, Riemann D, Hohagen F. Test-retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *J Psychosom Res* 2002;53:737-40.
59. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991;14:540-5.
60. Johns MW. Reliability and factor analysis of the Epworth Sleepiness Scale. *Sleep* 1992;15:376-81.
61. Joint National Committee on the Prevention, Evaluation, and Treatment of High Blood Pressure. The Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Bethesda, MD: U.S. Department of Health and Human Services, 2004. Report No.: NIH Publication No. 04-5230.
62. Patil SP, Schneider H, Schwartz AR, Smith PL. Adult obstructive sleep apnea: pathophysiology and diagnosis. *Chest* 2007;132:325-37.
63. Steinberg GG, Bleibtreu HK, Kurczynski TW, Martin AO, Kurczynski EM. Genetic studies in an inbred human isolate. In: Crow JF, Neel JV, eds. Proceedings of the Third International Congress of Human Genetics. Baltimore: Johns Hopkins University Press, 1967:267-90.
64. Abney M, McPeck MS, Ober C. Estimation of variance components of quantitative traits in inbred populations. *Am J Hum Genet* 2000;66:629-50.
65. Roberts RE, Roberts CR, Chen IG. Impact of insomnia on future functioning of adolescents. *J Psychosom Res* 2002;53:561-9.
66. Roehrs T, Roth T. Sleep, sleepiness, and alcohol use. *Alcohol Res Health* 2001;25:101-9.
67. Roehrs T, Roth T. Caffeine: sleep and daytime sleepiness. *Sleep Med Rev* 2008;12:153-62.

68. van den Berg JF, Knvistingh Neven A, Tulen JH, et al. Actigraphic sleep duration and fragmentation are related to obesity in the elderly: the Rotterdam Study. *Int J Obes (Lond)* 2008;32:1083-90.
69. Trento M, Broglio F, Riganti F, et al. Sleep abnormalities in type 2 diabetes may be associated with glycemic control. *Acta Diabetol* 2008;45:225-9.
70. Bonnet MH, Arand DL. Clinical effects of sleep fragmentation versus sleep deprivation. *Sleep Med Rev* 2003;7:297-310.
71. Tasali E, Leproult R, Ehrmann DA, Van Cauter E. Slow-wave sleep and the risk of type 2 diabetes in humans. *Proc Natl Acad Sci U S A* 2008;105:1044-9.
72. Stamatakis KA, Punjabi NM. Effects of sleep fragmentation on glucose metabolism in normal subjects. *Chest* 2010;137:95-101.