Original Contribution

Longitudinal Study on Poor Sleep and Life Dissatisfaction in a Nationwide Cohort of Twins

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Life satisfaction and quality of sleep are important, related components of subjective well-being and general health. However, no earlier investigation is known to have tested the direction of the temporal relation between poor sleep and diminished life satisfaction, including simultaneous examination of shared genetic influences. These features were examined in the present study of a nationwide cohort of 18,631 same-sex Finnish twins with repeated measurements of life satisfaction, sleep quality, and several potential confounders within an interval of 6 years (1975 and 1981). Most individuals (59%) with new-onset life dissatisfaction had experienced suboptimal sleep at baseline. Poor sleep predicted a consistent pattern of life dissatisfaction (odds ratio = 2.1, 95% confidence interval: 1.7, 2.7 from logistic regression on individuals; odds ratio = 3.0, 95% confidence interval: 1.7, 5.3 from conditional logistic regression on twin pairs discordant for life dissatisfaction), whereas life dissatisfaction did not consistently predict poor sleep. There was substantial heritability for both traits, but their shared genetic component was relatively weak, as indicated by genetic correlations of 0.21 for men and 0.27 for women in a multivariate genetic model. This finding is consistent with the hypothesis that poor sleep may have direct effects on the brain, emotions, and mood.

cohort studies; depression; incidence; life; personal satisfaction; sleep

Abbreviations: CI, confidence interval; OR, odds ratio.

Life satisfaction is a component of subjective well-being (1) and an indicator of psychological functioning (2). A decreased level is associated with poorer general health, disability, and mental distress and with an increased prevalence of smoking, heavy drinking, and physical inactivity (3). It is predictive for adverse health outcomes, including long-term mortality (4), suicide (5), and depressive symptoms (6). The latter link has great public health relevance because depression is one of the most important mental disorders worldwide, with a high prevalence and elevated mortality and disability (7). Estimates of the prevalence of life dissatisfaction in the general population vary. Some 12% of Finnish adults were dissatisfied with their life in the 1980s according to the life satisfaction scale (6, 8), while about 5% of US adults reported in 2005 that they were dissatisfied with their lives, a response based on a single question (3).

Sleep is an important contributor to general health and well-being. Sleeping 7 or 8 hours a night seems optimal for health, while both shorter and longer sleep predict morbidity and mortality (9, 10). A substantial proportion (18%–25%) of the population experiences insufficient or poor quality of sleep (11–13). Of various medical conditions, psychiatric disorders account for the largest diagnostic group of patients with sleep problems (14).

Associations between diminished life satisfaction and poor or insufficient sleep have been reported in a few cross-sectional and retrospective studies (3, 15, 16). In a British population, sleep duration of as many as 9 hours was positively associated with quality of life (13). In other studies, sleep satisfaction rather than duration was found to predict greater quality of well-being (17), well-being was more strongly related to the quality than the quantity of sleep.
sleep (18), and a dose-response relation was found between severity of insomnia at baseline and life dissatisfaction at follow-up among adolescents (2). However, few prospective longitudinal studies have explored whether life dissatisfaction predicts poor sleep or whether instead poor sleep predisposes for dissatisfaction in life.

Significant hereditary effects have been found on both sleep length and subjective sleep quality or sleep disturbance in previous studies (19, 20). So far, little information on heritability of life satisfaction is available. Genetic and environmental influences remained stable with 3 consecutive measurements of life satisfaction among Finnish adult twins, with additive genetic factors accounting for 25%–30% of the variance on each occasion and more than 50% of the covariance attributable to genetic factors (21). However, the role of shared genetic factors under the simultaneous or consecutive appearance of poor sleep and life dissatisfaction has not been studied to our knowledge.

Thus, no earlier investigation has tested the direction of the longitudinal relation between life dissatisfaction and nonoptimal sleep while simultaneously examining shared genetic influences. Further gaps in the evidence, such as whether it is length or quality of sleep that matters, or the role of several confounding factors such as sex, age, health behaviors, somatic illnesses, and stressful life events (3, 15, 20), also need to be taken into account.

Here, we investigated the direction of association between life dissatisfaction and nonoptimal sleep in a nationwide cohort of adult twins with measurements of life satisfaction, sleep quality, and sleep length within an interval of 6 years. We further explored the causal nature of those associations by using a matched case-control design. Finally, we tested whether shared genetic influences explained the associations between the 2 traits.

MATERIALS AND METHODS

Sample

The Finnish Twin Cohort was compiled from the Central Population Registry consisting of all same-sex twin pairs born in Finland before 1958 and with both co-twins alive in 1974 (13,888 pairs of known zygosity) (22). The project was accepted by the ethical committee of the University of Helsinki. Zygosity was determined by an accurate, validated questionnaire in 1975 and 1981 (22, 23).

Of the 22,220 persons replying to both the 1975 and 1981 surveys, we excluded those who were retired as of 1975 because of chronic disease or work disability, were unemployed, used hypnotics or tranquilizers more than 10 days during the past year, or performed night work (n = 3,069). After exclusions, the sample included 19,151 persons altogether. The data were complete for all main variables in both surveys—life satisfaction, sleep length, and sleep quality in 1975 and in 1981—for a total of 18,631 persons (8,914 men, 9,717 women). Because the values for some of the confounders were missing, the multivariate analysis sample comprised 18,211 individuals (8,713 men, 9,498 women). The mean age at baseline was 33 (range, 18–95; standard deviation, 12.7) years. In the genetic modeling, we excluded subjects for whom data on their co-twin were missing (n = 4,837) or twins of unknown zygosity (n = 830); the remaining data sample consisted of 12,964 individuals from 6,482 complete twin pairs, of which 2,168 were monozygotic (947 male, 1,221 female) and 4,314 were dizygotic (2,051 male, 2,263 female).

Measures

Life satisfaction. The questionnaire used to measure life satisfaction in 1975 and 1981 was a 4-item scale focusing on a feeling of loneliness, hardness of life, happiness, and anhedonia (4), modified for measuring the quality of life in Nordic countries (8). The complete scale ranges from 4 to 20 and can be categorized into 3 groups: “satisfied” (scores 4–6), “intermediate” (scores 7–11), and “dissatisfied” (scores 12–20). In the current study, it was dichotomized so that those respondents with a score of 12 or higher were coded as dissatisfied (encoded 1 for the statistical analysis) and those whose score was below 12 as satisfied (encoded 0), in a manner similar to that in previous reports from the Finnish Twin study (6).

Quality and length of sleep. We measured sleep in 1975 and 1981 with 2 self-reported variables: quality and length of sleep. Information on quality of sleep was obtained by asking, “Do you usually sleep well?” with 5 response alternatives: “well,” “rather well,” “rather poorly,” “poorly,” and “don’t know.” These responses were recoded into 3 categories so that sleeping well and rather well remained 2 separate categories, those sleeping rather poorly or poorly were combined into 1 category, and those who replied “don’t know” were coded as missing. In logistic regression analyses with sleep quality as an outcome, these variables were used as dichotomies, describing optimal (well or rather well) versus nonoptimal (poor or rather poor) sleep. Length of sleep was used here as a variable with 4 categories (less than 7, 7, 8, or 9 hours or more).

Confounders. We conducted multiple linear regression analyses to investigate potential confounders associated with life dissatisfaction and quality of sleep in 1981, based on available variables associated with these traits in earlier studies. On the basis of those analyses, the covariates to be adjusted for included sex, age, marital status (0 = married/cohabiting; 1 = single/living alone), education (number of years of school), somatic disease (e.g., diabetes mellitus, high blood pressure, angina pectoris, cardiac failure, stroke, chronic bronchitis, emphysema, asthma, allergy, urcharia, gastric ulcer, cholelithiasis, gout, or varicose veins: 0 = none, 1 = at least 1 chronic condition), life events (sum score of the 21-item Holmes-Rahe life event inventory) (24), smoking behavior (0 = never, 1 = occasional, 2 = former, 3 = current smoker) (25), alcohol consumption (binge drinking: yes/no) (26), and physical activity (0 = sedentary, 1 = moderate, 2 = active) (27). Information on these covariates was collected in 1975, except for life events, which were assessed in 1981.

Statistical analyses

In the logistic regression analyses, the subjects were considered as individuals, but we controlled for twinship. The
models tested the strength and significance of predictive associations between life dissatisfaction and sleep quality or length in 1975 and 1981 so that good life satisfaction ("satisfied") as well as good sleep quality ("sleeping well") and 8 hours of sleep constituted the reference groups (odds ratio (OR) = 1.00). Those "dissatisfied" already in 1975 were excluded from the analysis of incidence of life dissatisfaction in 1981; similarly, those reporting rather poor or poor sleep in 1975 were excluded from the analysis of incidence of poor sleep in 1981. Odds ratios with 95% confidence intervals were computed by adjusting for age and sex. We also tested sex and age interactions for each predictor variable. If the interaction by sex was significant, the analyses were also conducted separately for men and women; otherwise, data for both sexes were combined. Multiple logistic regression models were used to adjust for the confounders listed above. Because observations of twins within twin pairs may be correlated, we used robust estimators of variance and the cluster option in Stata software when estimating standard errors.

Because the data were for twins, there was a unique opportunity to explore the causal nature of the associations between predictors and outcomes. To test whether life dissatisfaction predicts poor sleep independently of within-family confounders, we identified all twin pairs, disregarding zygosity, discordant for life dissatisfaction in 1981 as matched cases and controls sharing the same early environment and familial background. Similarly, to test whether poor sleep predicts life dissatisfaction, we identified all twin pairs discordant for poor sleep in 1981 as matched cases and controls. We first used the McNemar test as an unadjusted test, and then we applied conditional multiple logistic regression to adjust for confounding variables not matched for in the design. All logistic and conditional logistic regression analyses were conducted by using the Stata statistical package, version 9 (29). The level of statistical significance was considered as a P value of less than 0.05.

To monitor for genetic influences on the analyzed traits, we compared the risk of life dissatisfaction and poor sleep for discordant monozygotic and dizygotic twin pairs (30). We then computed the cross-twin intratrait, the intratwin cross-trait, and the cross-twin cross-trait correlations separately for the monozygotic and dizygotic pairs. Finally, we used univariate and multivariate twin models to explore whether the observed associations could be ascribed to underlying genetic factors in common, that is, shared by both phenotypes (31, 32). The greater resemblance of monozygotic versus dizygotic pairs regarding this association, as indicated by cross-twin cross-trait correlations, is formally modeled by decomposing the phenotypic correlation into correlations between genetic and environmental components of life dissatisfaction and sleep quality. The modeling was based on standard Mx (33) scripts obtained from the GenomEUtwin Mx website (http://www.psy.vu.nl/mxbib/).

### RESULTS

#### Life dissatisfaction as a predictor of poor sleep quality

We first explored the temporal relation between life dissatisfaction in 1975 and quality of sleep in 1981 (Table 1). The analyses of incidence were performed by using multiple logistic regressions adjusted for sex, age, and other confounders (marital status, education, somatic disease, life events, smoking behavior, alcohol consumption, and physical activity).

We found an association between life dissatisfaction and subsequent poor sleep quality among those who slept well at baseline (n = 8,866) (adjusted OR = 1.20, 95% confidence interval (CI): 1.03, 1.40), with a significant interaction...
between sex and life dissatisfaction ($P = 0.03$), so that the association was significant for men ($OR = 1.41, 95\% CI: 1.13, 1.77$) but not for women ($OR = 1.02, 95\% CI: 0.83, 1.27$). However, the conditional logistic regression analysis did not support the hypothesis that life dissatisfaction predicts future sleep problems ($OR = 0.93, 95\% CI: 0.63, 1.38$) (Table 1).

**Poor sleep quality as a predictor of life dissatisfaction**

Next, we examined the predictive relation between quality of sleep measured in 1975 and life dissatisfaction in 1981. Individuals reporting poor sleep quality in 1975 had significantly elevated risks of being dissatisfied in 1981. Furthermore, we found a dose-response relation between baseline sleep quality and subsequent incidence of life dissatisfaction. Those sleeping rather poorly or poorly demonstrated a 2-fold risk of life dissatisfaction (adjusted $OR = 2.11, 95\% CI: 1.68, 2.65$) (Table 2). Adding sleep length as a covariate into the model did not alter these estimates. Sleep length itself had only a modest effect, so that the odds of subsequent life dissatisfaction were slightly higher for those who slept fewer than 7 hours per night in 1975 compared with those with an 8-hour sleep ($OR = 1.31, 95\% CI: 1.09, 1.58$ when sleep quality was included simultaneously in the model). Among twin pairs discordant for life dissatisfaction in 1981, poor quality of sleep remained a significant predictor of the incidence of life dissatisfaction ($OR = 2.11, 95\% CI: 1.68, 2.65$) (Table 2).

**Genetic influences on sleep quality and life dissatisfaction**

Finally, we tested for the presence of genetic influences on the phenotypic associations between life dissatisfaction and poor sleep quality. We first examined the phenotypes separately, then concomitantly, by conducting univariate and multivariate Mx twin models (33).

Genetic components for both traits seemed to be rather stable over time. For sleep quality, slightly higher heritability estimates were obtained for women at baseline (1975) than 5 years later (1981), and the estimates for life dissatisfaction were similarly higher for the younger men and women than for the older ones. Because the most parsimonious univariate model was that including only estimates for additive heritability and estimates for unique environmental effects with different variance components and prevalences for men and women, heritability estimates were computed separately by sex, including age as a covariate. For men, the heritability estimates for sleep quality were 33% in 1975 and 39% in 1981; for women, they were 53% in 1975 and 39% in 1981. Regarding life dissatisfaction, the most parsimonious model was the additive genetic–unique environmental model, with the same variance components and prevalences for both sexes and without a significant age effect, leading to heritability estimates of 42% in 1975 and 29% in 1981.

To observe whether covariation between poor sleep and life dissatisfaction could be accounted for by shared genetic or environmental correlations, the multivariate analyses based on Cholesky decompositions were conducted by using Mx (33). All 4 phenotypes—sleep quality in 1975 and 1981, as well as life dissatisfaction in 1975 and 1981—were taken simultaneously into consideration. On the basis of results from the univariate models, the additive genetic–unique environmental multivariate models, including age effect, were fitted separately for men and women. The heritability estimates for phenotypes in 1981 among men dropped quite dramatically, whereas, among women, these values increased slightly compared with those obtained
from the univariate models. As expected, there were relatively strong genetic contributions to the stable components of both sleep quality and life dissatisfaction. The correlations of the genetic components over time were $r_g = 0.78$ for quality of sleep in 1975 and 1981 among men and $r_g = 0.87$ among women; for life dissatisfaction, they were $r_g = 0.93$ and $r_g = 0.90$, respectively (Figure 1). In contrast, the evidence for a shared genetic component between concomitantly or consecutively measured poor sleep quality and life dissatisfaction was scanty. We did not observe evidence for shared genetic influences on the cross-twin cross-trait correlations of sleep quality in 1975 and life dissatisfaction in 1981 (0.04 and 0.17 for monozygotic and −0.04 and 0.02 for dizygotic male twins; 0.07 and 0.10 for monozygotic and 0.06 and 0.10 for dizygotic female twins, respectively).

Furthermore, in line with these preliminary findings, the multivariate models produced only modest estimates of the genetic correlations between sleep quality in 1975 and life dissatisfaction in 1981: $r_g = 0.21$ for men and $r_g = 0.27$ for women, respectively.

**DISCUSSION**

Our results showed a predictive relation between poor sleep quality and subsequent incident life dissatisfaction, whereas the existence of a converse relation was not supported. Our finding was statistically robust so that the risk did not vanish when confounding factors, such as somatic disease, alcohol consumption, or life events, were considered, and it was also observed within discordant twin pairs. We recognize as a limitation that information on most confounders was collected at the baseline but not the follow-up survey, while some of these confounders could have changed between those 2 assessments. One additional confounding factor could have been sleep length, because insufficient sleep duration can be either a cause or a consequence of an individual notion of poor sleep quality. However, adding sleep length as a covariate into the analysis did not alter the estimates; furthermore, sleep length itself had only a modest effect on the incidence of life dissatisfaction.

We used the scale for life satisfaction and applied it as a dichotomy, which has previously been found to detect moderate or severe depression with 87% sensitivity and 88% specificity (6). Although providing an approximation of depressed mood, the scale may be less indicative of other features encountered in major depression, such as performance impairment or somatic symptoms such as appetite change, weight loss, or insomnia (34). A number of earlier studies have shown that poor sleep presents a risk of depression in the adult population (35, 36) as well as for adolescents (37), with an estimated risk of 2–4 in most studies. However, many of these studies lacked population-based, systematic ascertainment. In addition, since age itself has a well-known effect on sleep, results for the aging population or young adults should be generalized to a broad adult population with caution. The direction of risk between depression and poor sleep was investigated retrospectively among 1,014 adolescents; prior depression was not associated with insomnia, whereas prior insomnia was associated with depression, with a hazard ratio risk estimate of 3.8 (95% CI: 1.6, 8.6) (37).

Although in concordance with those findings, the present results extend them in several important ways. Our study was based on a systematically collected, nationwide cohort, whereas the previous study excluded cases without health insurance or those with traditional insurance. Furthermore, our sample included only those individuals from the adult population who were followed longitudinally, allowing for more precise examination of the temporal relation. Finally, our sample comprising same-sex twins allowed discrimination between the shared or distinctive genetic factors underlying these examined features.
Life satisfaction covers several domains of health and well-being (3, 38) and is associated with many mental, physical, and social indicators. The life satisfaction scale (4) used in the current study included domains for measuring quality of life (8). These domains seem to capture more the mental and social than the physical indicators of health and have a high correlation with depressiveness assessed by the Beck Depression Inventory (6). Regarding poor sleep in relation to other domains, it has been associated with impaired somatic functioning among adolescents (2), and insomnia has been related to dissatisfaction particularly with social contacts among the elderly (16). Sleep satisfaction was also associated with the Quality of Well-Being Scale, which includes information on mobility and on physical and social activity (39). In the working-age population, poor sleep has been associated with work-related life satisfaction, mediated by poor physical and psychological health, absenteeism, and problems with work performance and personal relationships (40). Finally, a meta-analysis on insomnia and heart diseases (41) suggested that subjective sleep complaints may be part of a larger syndrome including poor health and depression, or they may be related to continual stressors, reduced slow-wave sleep, and autonomic dysfunction, which increase the risk of heart problems.

The heritability estimates of both life dissatisfaction and sleep quality seemed to be stable over time and, in general, to accord with earlier findings (19, 21, 42, 43). The values for women and men differed, however, so that the genetic component was higher for women (37%–36% for life dissatisfaction, $r_g = 0.90$; 53%–41% for sleep quality, $r_g = 0.87$) than for men (30%–15% for life dissatisfaction, $r_g = 0.93$; 21%–22% for sleep quality, $r_g = 0.78$). This finding might reflect the effect of endocrine factors such as sex steroids on central nervous system functions or neurotransmitter systems (44). Many genes involved in mechanisms regulating homeostatic or circadian components of sleep or related to mood disorders have been shown to be under transcriptional modification by sex hormones (45–48). Moreover, a number of psychiatric traits or features are different regarding their prevalence and heritability estimates for men and women. For example, in a recent study, heritability of major depression was found to be clearly higher among women (42%) than among men (29%), and the effect of some genetic risk factors for major depression was suggested to be sex specific (49).

Interestingly, only a weak genetic correlation was found between poor sleep quality and life dissatisfaction at the different time points, indicating that most of the genetic factors contributing to these traits are unique in regard to the other feature. Thus, finding a temporal relation between poor sleep and subsequent life dissatisfaction is likely not to represent solely the dynamic reflections of the same underlying genetic factors. Rather, it is likely to illustrate more causal mechanisms underlying the neurophysiologic effect of poor sleep on brain function, and the experience of feelings of dissatisfaction in life.

The neurobiology of disturbed sleep in depression is well documented, with disturbances of the electrophysiological sleep architecture (50) as well as functional deviations in the different brain regions (51) of individuals with depressive disorder. In the current study, only subjective information on sleep quality was available. The role of objective and subjective sleep measures in depression is controversial (52). In healthy controls, subjective experiences with poor sleep quality were found to be associated with electrophysiological architecture of sleep as well, that is, a decrease in slow-wave sleep and an increase in number of awakenings (53). Interestingly, these features are also encountered in depression (54). One might hypothesize that these kinds of changes in sleep architecture could present the neurophysiologic measures for the experience of poor sleep that we found to be temporally related to subsequent life dissatisfaction. Eventually, studies on a large longitudinal cohort with detailed electrophysiological data will be needed to test this hypothesis.

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