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Effects of Cognitive Behavioral Therapy for Insomnia on Sleep-Related Cognitions among Patients with Stable Heart Failure

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Abstract

Objective/Background—Cognitive behavioral therapy for insomnia (CBT-I) improves insomnia and fatigue among chronic heart failure (HF) patients, but the extent to which sleep-related cognitions explain CBT-I outcomes in these patients is unknown. We examined the effects of CBT-I on sleep-related cognitions, associations between changes in sleep-related cognitions and changes in sleep and symptoms after CBT-I, and the extent to which cognitions mediated the effects of CBT-I.

Participants—Stable New York Heart Association Class II-III HF patients (total $n = 51$; $n = 26/51.0\%$ women; $\bar{M}$ age = 59.1 ± 15.1 years).

Methods—HF patients were randomized in groups to group CBT-I ($n = 30$) or attention control (HF self-management education) ($n = 21$) and completed actigraphy, the Insomnia Severity Index, Pittsburgh Sleep Quality Index, Dysfunctional Beliefs and Attitudes about Sleep (DBAS) and Sleep Disturbance Questionnaires (SDQ), and self-reported fatigue, depression, anxiety, and sleepiness (baseline, immediately after treatment, six months). We used mixed effects modelling, mediation analysis with a bootstrapping approach, and Pearson correlations.

Results—There was a statistically significant group by time effect on DBAS. DBAS mediated the effects of CBT-I on insomnia severity and partially mediated CBT-I effects on fatigue. Improvements in dysfunctional cognitions were associated with improved sleep quality, insomnia

severity, sleep latency and decreased fatigue, depression, and anxiety, with sustained effects at 6 months.

Conclusions—Improvement in dysfunctional sleep-related cognitions is an important mechanism for CBT-I effects among HF patients who are especially vulnerable to poor sleep and high symptom burden.

Keywords

heart failure; insomnia; cognitive behavioral therapy; sleep; fatigue; depression; beliefs; attitudes; perceptions; actigraphy; sleep quality

Introduction

Over five million Americans (Go et al., 2014) and many more throughout the world suffer from chronic heart failure (HF), a condition associated with excessive morbidity, mortality, comorbidity, and symptom burden. Insomnia is prevalent in HF patients, occurring in as many as 25–56% of those with chronic HF (Brostrom, Stromberg, Dalstrom, & Fridlund, 2004; Gau, Chen, Wu, Lin, & Chao; Redeker et al., 2010; Redeker & Stein, 2006). Insomnia is closely associated with fatigue, depressive symptoms, daytime sleepiness and decrements in self-reported and objective functional performance (Redeker et al., 2010) – important concerns for these patients. Insomnia predicted incident HF and death in a population-based study (Laugsand, Strand, Platou, Vatten, & Janszky, 2014) and adverse cardiac events among patients who already had HF (Kanno et al., 2016).

The negative effects of insomnia on critical outcomes among HF patients suggest the importance of behavioral treatment, such as cognitive behavioral therapy for insomnia (CBT-I), especially given the negative consequences of hypnotic medications (Andrews, Coviello, Hurley, Rose, & Redeker, 2013). Indeed, CBT-I had large effects on insomnia severity, sleep efficiency, and fatigue among patients with stable HF in a pilot efficacy trial (Redeker et al., 2015). However, little is known about the cognitive mechanisms that may explain these effects.

Although sleep disturbance is often attributed to HF exacerbations (e.g. fluid overload), insomnia occurs even in stable patients on prescribed evidence-based HF medical therapy and, contrary to common clinician perceptions, was not explained by sleep disordered breathing or severity of HF among stable patients on standard evidence-based HF treatment (Redeker et al., 2010). HF patients report perpetuating factors for insomnia including increased time in bed awake (Redeker & Stein, 2006), perceptions of insufficient sleep, frequent napping (Brostrom et al., 2004; Erickson, Westlake, Dracup, Woo, & Hage, 2003; Redeker & Stein, 2006), and use of the TV as background sound to help them sleep (Andrews et al., 2013). There were also positive associations between sleep hygiene behavior and sleep quality among HF patients (Riegel et al., 2012). While data from a qualitative study (Andrews et al., 2013) suggest that worries and misperceptions about sleep are common and may contribute to chronic insomnia, dysfunctional sleep related cognitions (i.e., perceptions, attitudes, beliefs, thoughts) have not been systematically examined in this population.

Dysfunctional sleep-related cognitions are closely related to insomnia among patients with primary insomnia and insomnia associated primarily with psychiatric disorders (Arnedt et al., 2007; Carney & Edinger, 2006; Carney, Edinger, Manber, Garson, & Segal, 2007; Carney et al., 2010; Cronlein et al., 2014; Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001; Fairholme & Manber, 2014; Morin, Blais, & Savard, 2002). Improvements in sleep-related cognitions are associated with improvements in insomnia, depressive symptoms (Sunnhed & Jansson-Frojmark, 2014), and fatigue among patients with anxiety and depressive disorders (Fairholme & Manber, 2014), while CBT-I had direct short term effects on improvement in dysfunctional sleep-related cognitions among patient with primary insomnia (Edinger et al., 2001; Montserrat Sanchez-Ortuno & Edinger, 2010; Morin et al., 2002; Yamadera et al., 2013). However, few studies, if any, have examined the direct effects of CBT-I on cognitions or the extent to which dysfunctional cognitions mediate the effects of CBT-I on insomnia and daytime symptoms among patients with life threatening medical comorbidity, such as HF. Therefore, the aims of this study were to (1) evaluate the effects of CBT-I on sleep-related beliefs and cognitions; (2) investigate the associations between changes in sleep-related beliefs and cognitions and changes in insomnia, sleep characteristics, and daytime symptoms; and (3) determine the extent to which sleep related beliefs and cognitions mediated the effects of CBT-I on insomnia and fatigue

METHODS

Design

We conducted a pilot randomized controlled trial (RCT) of CBT-I among patients with stable HF and insomnia in which participants were randomized in groups of 4–5 to four weeks of bi-weekly CBT-I in a group format with a telephone call on the intervening weeks (total eight weeks) or an attention control condition (HF self-management education) delivered in the same format (Redeker et al., 2015). We previously reported full details of the protocol and its feasibility, acceptability, and preliminary efficacy among HF patients (Redeker et al., 2015). We obtained human subjects' approval, and all participants provided written informed consent.

Setting & Sample

We conducted the study in the northeastern United States and recruited HF participants from a tertiary care HF program and the surrounding community. Included participants were aged 18 years of age or older, lived at home, were cognitively intact by clinical impression and had New York Heart Association Classification II–III HF, and an Insomnia Severity Index (ISI) > 7, indicative of at least mild chronic insomnia (Bastien, Vallieres, & Morin, 2001). We excluded individuals if they had untreated sleep disordered breathing (Apnea Hypopnea Index ≥ 10) as assessed by an ambulatory sleep study [Apnea Risk Evaluation System: ARES, Watermark Medical, Inc.] or narcolepsy, performed night or rotating shift work, or had untreated restless legs syndrome or end-stage renal failure requiring dialysis. Patients with mild sleep apnea have benefitted from CBT-I (D. J. Buysse et al., 2011). Potential participants were excluded if they had seizure disorders or neurological or musculoskeletal disorders that restricted movement of the non-dominant arm because of the possible confounding effects on wrist actigraph recordings. We did not exclude participants who were

taking prescribed or over the counter hypnotic medications (Redeker et al., 2015) because hypnotic taper was an optional component of the intervention. Participants using positive airway pressure (PAP) treatment for sleep apnea were included in the study if adherent by self-report for 6 hours/night for at least 6 nights per week.

The sample size of 51 ensured detection of previously observed correlations between the Dysfunctional Beliefs and Attitudes about Sleep (DBAS) scale score and sleep quality as assessed by the Pittsburgh Sleep Quality Index – (PSQI) ($r = 0.51$), anxiety ($r = 0.46$), and depression ($r = 0.41$) in non-clinical populations with powers of 86.2% and higher (Gadam, 2015). This sample size has a power of 78.4% to detect a group by time interaction (Noncentrality parameter = 7.68) on DBAS at a significance level of 0.05.

Procedures

We obtained written informed consent and screened potential participants to determine eligibility (Redeker et al., 2015). Participants completed two weeks of baseline evaluation, including twenty four-hour wrist actigraphy, sleep diaries, and study questionnaires. Participants were randomized to either CBT-I or the attention-control condition in groups of four or five, but not informed of the group assignment until the group meeting, and then participated in either condition. Measurements were repeated two weeks after group participation, and the self-report measures were completed again six months after study participation.

A clinical psychologist certified in sleep medicine provided the CBT-I in 4 bi-weekly sessions over an eight-week period with telephone calls on intervening weeks. The CBT-I included sleep hygiene, cognitive therapy, stimulus control, sleep restriction, progressive muscle relaxation, and optional advice on hypnotic tapering (Redeker et al., 2015). The attention control group included HF self-management education, based on the American Association of Heart Failure Nurses' Fight against HF Handbook (American Association of Heart Failure Nurses, 2008) led by an advanced practice nurse (APRN) in the identical format to the CBT-I group as control for time and attention. Sleep hygiene information was provided to this group, but is not the active ingredient of CBT-I (McCrae et al., 2006; Morin et al., 2006).

Variables and Measures

Clinical and demographic variables—We elicited demographic and clinical characteristics. We used the Charlson Comorbidity Index (Charlson, Pompei, Ales, & McKenzie, 1987) and the New York Heart Association Functional Classification to describe clinical characteristics of the sample.

Sleep Characteristics—To elicit the multi-dimensional subjective and objective attributes of sleep (quality, duration, continuity, latency) and patient perceptions (sleep quality, insomnia severity), we obtained actigraphy and self-report sleep measures. The Pittsburgh Sleep Quality Index (PSQI) (D. J. Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) assesses sleep efficiency, duration, latency and global sleep quality and is reliable and valid (D. J. Buysse et al., 1991; D. J. Buysse et al., 1989). Global sleep quality ranges from

0–21 (higher score indicates poorer sleep quality and a score > 5 characterizes poor sleep quality). The PSQI distinguishes “good and poor” sleep, with sensitivity of 89.6% and specificity of 86.5% distinguishing “good” vs. “poor” sleep (D. J. Buysse et al., 1989). We elicited time in bed (time from going to bed to getting up in the morning); time asleep; and sleep latency (minutes to falling asleep). From these data, we computed sleep efficiency [(sleep time/time in bed) X 100]. We used the self-reported “raw” data, rather than the component scores, as this calculation summarizes the data that would be obtained by individual nights of diary data. The PSQI components were associated with sleep diary parameters (Grandner, Kripke, Yoon, & Youngstedt, 2006).

We used the Insomnia Severity Index (ISI) (Bastien, Vallieres, & Morin, 2001) to evaluate insomnia severity. The ISI is internally consistent (0.74 – 0.88) (Bastien et al., 2001; Blais, Gendron, Mimeault, & Morin, 1997) and sensitive to treatment (Bastien et al., 2001; Blais et al., 1997; Smith & Trinder, 2001) with possible scores ranging from 0–28. Cronbach’s alpha was .83 in the current sample.

We measured objective sleep characteristics with the Respironics Minimitter Actiwatch AW2, a wrist-worn accelerometer. Correlations between actigraphy and polysomnographic (PSG) sleep variables are between 0.82–0.98 (sleep efficiency) and 0.90–0.97 (sleep duration) in normal sleepers (Benson et al., 2004; Blood, Sack, Percy, & Pen, 1997; Edinger, Means, Stechuchak, & Olsen, 2004; Kushida et al., 2001). Actigraphy corresponded with PSG on sleep efficiency, awakenings, wake after sleep onset, and total sleep time among people with insomnia (Lichstein et al., 2006) and is sensitive to changes over time and treatment (Littner et al., 2003).

Daytime Symptoms—We measured common daytime symptoms experienced by HF patients and often related to insomnia (i.e., fatigue, excessive daytime sleepiness, anxiety, and depression). The Multi-dimensional Assessment of Fatigue Index (MAF) elicits severity, distress, degree of interference and timing of fatigue (Belza, 1990) and has concurrent ($r = 0.84$) and divergent ($r = -0.62$) validity, and internal consistency ($\alpha = 0.84$) in HF patients (Redeker, 2006).

The Epworth Sleepiness Scale (ESS) (Johns, 1991) was used to measure self-reported daytime sleepiness. It is reliable in HF patients (coefficient $\alpha = 0.87$) (Redeker, 2006). The Center for Epidemiological Studies Depression Scale (CESD) (Devins & Orme, 1985) was used to measure depressive symptoms. Cronbach’s alpha was 0.83 in HF patients (Redeker, 2006). The 40-item Spielberger State Inventory Form Y was used to measure state anxiety. Test-test reliability exceeds 0.7 (Spielberger, 1983).

Sleep-Related Cognitions—We used two measures to elicit sleep-related cognitions. The Dysfunctional Beliefs and Attitudes about Sleep Scale (Carney et al., 2010; Morin, Vallieres, & Ivers, 2007) was used to measure maladaptive beliefs about sleep. The items are measured on a 0–10-point numeric scale. The scale has acceptable internal consistency and stability (Morin et al., 2007), and the alpha coefficient was 0.89 in this study. A higher score reflects higher dysfunctional beliefs and attitudes about sleep.

The Sleep Disturbance Questionnaire (SDQ) is a 12-item scale designed to evaluate beliefs about the sources of insomnia (Espie, Inglis, Harvey, & Tessler, 2000). The SDQ includes four factors that indicate attributions related to insomnia (restlessness/agitation, mental over-activity, consequences of insomnia, and lack of sleep readiness). The SDQ was validated in populations with chronic insomnia (Espie et al., 2000; Smith & Trinder, 2001; Violani, Devoto, Lucidi, Lombardo, & Russo, 2004). Cronbach's alpha was 0.87 at baseline in the current study.

Data analysis

Actigraph data were downloaded into Actiware v. 5 software (Respironics Minimitter, Inc.). The data were visually reviewed, and "lights out" and "lights on" times were determined from event marker recordings. If event markers were missing, we used the diary recordings and visual analysis of the data to determine the major sleep interval (lights out time to arising time). We computed nocturnal sleep duration, efficiency, and sleep latency for each day using the standard algorithm and a medium sensitivity setting on the software. Although sleep latency is often not a stable variable when measured with actigraphy, its association with severity of HF, measured with the New York Heart Association Functional Classification in our previous work (unpublished data) suggests its usefulness in this population. Descriptive statistics were computed for the actigraph sleep variables over both of the 2-week intervals of data collection. We did not collect actigraphy data at follow-up time 2 (six months).

Data were committed to an MS Access database. We used logical data checks to identify outliers and data errors after importing the data into SAS. Descriptive statistics were analyzed for all variables to examine frequency distributions of categorical variables and normal distributions for continuous variables.

Baseline equivalence on demographic characteristics and potential confounding variables was assessed between the two groups to determine the success of randomization using t-tests and Wilcoxon tests. Variables differing by group assignment were included in the models as covariates.

We evaluated the intervention effects over time [baseline, follow-up time 1 (immediately after treatment) and follow-up time 2 (6 months)] on the DBAS and the SDQ using mixed effects models with random intercepts and autoregressive correlation structures to address within-subjects' changes. The group-by-time interactions were tested after controlling for age and comorbidity. We used the Kolmogorov-Smirnov test to evaluate normality of residuals.

We calculated changes in the DBAS, SDQ, sleep quality, self-reported and actigraph-recorded sleep characteristics, and daytime symptoms from baseline to follow up times 1 and 2. The cross-sectional correlations of changes in the DBAS and SDQ (possible mediators) from baseline to time 1 and baseline to time 2 with the sleep measures and daytime symptoms were assessed with Pearson's correlation coefficients. To control for type I error inflation, we calculated the false discovery Rate (FDR), accounting for multiple tests using PROC MULTTEST, SAS version 9.3 (Glickman, Rao, & Schultz, 2014). We

performed six simultaneous tests for hypothesized associations with changes in sleep characteristics and four simultaneous tests for hypothesized associations with daytime symptom changes from baseline to times 1 and 2.

We used a traditional approach (Baron & Kenny, 1986) to examine the extent to which sleep-related beliefs and cognitions mediated the treatment effects on insomnia and fatigue, given that CBT-I improved insomnia and fatigue in this sample (Redeker et al. 2015). Direct and Indirect (Mediation) effects and 95% confidence intervals were estimated based on two regression models of changed mediators and outcomes during follow-up time 2 using 2,000 bootstrap samples (MacKinnon, 2008).

RESULTS

We previously reported details of subject accrual, randomization and retention for the overall study (Redeker et al., 2015). Of the 52 enrolled participants who were randomized to attention control ($n = 29$) or CBT-I ($n = 19$), we previously reported data on 48 participants at baseline and time 1 (immediately post treatment). For the present analysis, one additional participant was missing baseline data on the DBAS or SDQ. Therefore 51 participants had usable baseline data for this report ($n = 30$, CBT-I condition; $n = 21$ attention control); 28 and 22 in the CBT-I group had complete data (times 1 and 2, respectively), and 19 and 13 in the attention-control. Two participants in the attention-control condition were not eligible for follow-up at six months because they obtained CBT-I after completing the control condition.

Clinical and demographic characteristics of the sample and the treatment groups are presented in table 1. Although there was a slightly higher age and percentage of women in the CBT-I group, the difference between groups was not statistically significant. There was higher comorbidity in the CBT-I compared to the attention control group (Wilcoxon, $p = .0213$).

Table 2 includes the means and standard deviations of the dysfunctional beliefs and attitudes about sleep scale (DBAS) and sleep disturbance questionnaire (SDQ) scores over three time points (baseline and two follow-up periods). The coefficients for the group by time interactions and covariates estimated from the mixed effect models are presented in Table 3. There was a statistically significant group by time interaction ($p = .0072$) effect on DBAS, with a significant decrease in the CBT-I group (-0.600 ± 0.190) compared to the attention control group (HF Self-management) in which there was no decrease (0.242 ± 0.239). A statistically significant decrease was observed in the SDQ in the CBT-I group (-0.350 ± 0.076), but the SDQ score at follow-up was not significantly different compared to the attention control group ($p = .1137$).

The correlation between the DBAS and the SDQ scores was $r = 0.556$ ($p < .0001$) at baseline, and these associations were similar and moderate to large at times 1 (immediately after CBT-I) ($r = 0.447$, $p = .0016$) and at time 2 (six months) ($r = 0.619$, $p < .0001$), while the correlation between changes in the DBAS and changes in the SDQ scores from baseline to time 2 was high ($r = 0.71$, $p < .001$). Decreases in both the DBAS and SDQ scores from baseline to time 1 and from baseline to time 2 were moderately and positively associated

with decreases in insomnia severity and improved self-reported global sleep quality (high PSQI = low sleep quality) with the greatest correlation for the time from baseline to time 2. (See Table 4). The associations between changes in the DBAS and Insomnia at time 1 and time 2 and with sleep quality at time 2 and the associations between changes in SDQ and sleep quality between baseline and times 1 and 2 were statistically significant based on the False Discovery Rate Analysis

Improvement in DBAS (decreased score) was associated with a non-significant increase in actigraph-recorded, but not self-reported, sleep duration and a statistically significant decrease in self-reported sleep latency over both time frames, with the highest correlations with sleep latency, insomnia severity, and sleep quality at time 2. Decrease in the SDQ score from baseline to time 1 was associated with increased self-reported sleep duration from baseline to time 1, and the change from baseline to time 2 was associated with sleep latency (See Table 4). Neither the DBAS nor the SDQ were associated with changes in self-reported or actigraph-recorded sleep efficiency (data not shown).

There were positive small-moderate correlations between decreases in the DBAS and SDQ and decreases in fatigue from baseline to time 1, with higher correlations that were statistically significant for the changes between baseline and time 2, compared to the changes from time 1 to time 2 (See Table 4). Decreases in the SDQ from baseline to time 1 had a statistically significant association with improvement in depression, and decreased SDQ had a moderate and statistically significant positive correlation with decreased depression from baseline to time 2. Changes in both the SDQ and DBAS had moderate associations with changes in anxiety at time 2. There was no association between changes in the SDQ or DBAS and changes in excessive daytime sleepiness. Figure 1 shows the scatter plots of changes in DBAS, SDQ, insomnia severity, and daytime symptoms for the entire sample from baseline to time 2 (six month) follow-up.

The distributions of the indirect (mediation) effects calculated from 2,000 bootstrap samples are shown in Figure 2. Dysfunctional beliefs and cognitions, as measured by the DBAS, significantly mediated the CBT-I effect on improving insomnia (Indirect effect = -0.47 , 95% CI = -0.93 , -0.06) and fatigue (indirect effect = -0.42 , 95% CI = -0.84 , -0.05), but DBAS only partially mediated the CBT-I effect on fatigue, and the direct effect remained statistically significant (direct = -0.98 , 95% CI = -1.22 , -0.20) in the mediational model. Sleep related attributions, as measured by the SDQ, did not mediate the treatment effects of CBT-I on insomnia severity (indirect $t = -0.19$, 95% CI = -0.54 , 0.03) or fatigue (indirect = -0.26 , 95% CI = -0.67 , 0.02).

DISCUSSION

The findings of this study suggest that CBT-I improves dysfunctional cognitions about sleep among patients with stable heart failure (HF) and that improvements in dysfunctional sleep-related cognitions explain improvements in insomnia severity and partially explain improvements in fatigue. Our results are important because they contribute to understanding the role of sleep-related cognitions as mechanisms for the effects of CBT-I among HF patients, a group that suffers from very significant health challenges, including a high

prevalence of insomnia and daytime symptoms (Redeker et al., 2010). Elucidation of the mediational effects of sleep-related cognitions in our study extends knowledge from previous studies that primarily considered associations between cognitions and sleep characteristics or the direct effects of CBT-I on cognitions (Arnedt et al., 2007; Carney & Edinger, 2006; Carney et al., 2007; Carney et al., 2010; Cronlein et al., 2014; Edinger et al., 2001; Fairholme & Manber, 2014; Montserrat Sanchez-Ortuno & Edinger, 2010; Morin et al., 2002; Sunnhed & Jansson-Frojmark, 2014; Yamadera et al., 2013).

Despite differences in the clinical characteristics between our sample and those of previous studies, the level of the DBAS score at baseline in both treatment groups was higher than the level ($M = 3.8$) suggesting clinically significant insomnia (Carney et al., 2010). The decrease of DBAS below the level of 3.8 in the CBT-I group at both follow-ups further underscores the clinically meaningful improvement in insomnia resulting from CBT-I in this sample (Redeker et al., 2015).

Decreases in the DBAS and SDQ scores were consistently associated with improvements in sleep quality and insomnia severity, but not associated with changes in sleep efficiency, despite statistically significant large, but clinically small, improvements in actigraph-recorded sleep efficiency resulting from CBT-I (Redeker et al., 2015). The lack of an association with sleep efficiency contrasts with the association found in a previous study of patients with primary sleep maintenance insomnia (Edinger et al., 2001), but this finding should be examined in a larger more fully powered study of patients with HF.

Although both the DBAS and SDQ scores improved in the CBT-I group compared to the attention control, the only statistically significant group by time effect was on the DBAS score. While the DBAS and SDQ measure different cognitive constructs [maladaptive beliefs about sleep (DBAS) and attributions about insomnia (SDQ)] (Espie et al., 2000), our data suggest that both are important to insomnia. The consistent improvements in the CBT-I group compared to the attention control, similarities in the associations between changes in DBAS and SDQ scores and changes in the sleep and symptom variables, and the close correlations between the DBAS and SDQ, suggest the need for further study of the effects of CBT-I on specific cognitions (including subscale scores of the DBAS and SDQ) in a study that is powered to evaluate these effects. This information may be useful in identifying the specific cognitions that should be targeted to improve outcomes among HF patients in order to refine the treatment. Given that the attention control condition included sleep hygiene education, these findings support the specific role of cognitive therapy as a component of CBT-I.

Although there were robust correlations between improvements in dysfunctional cognitions and improvements in fatigue, anxiety, and depression, especially at six months, we did not find a direct effect on anxiety or depression in our previous report obtained from this sample (Redeker et al., 2015). Our finding that cognitions only partially mediated the large CBT-I effects on fatigue suggests that other behavioral mechanisms or CBT-I components other than cognitive therapy (e.g. sleep restriction) may better explain the direct effect of CBT-I on fatigue. (Redeker et al., 2015).

The associations found between changes in cognitions and changes in symptoms may be explained in several ways. Improvement in symptoms may be a generalized response to improved sleep-related cognitions or may be directly related to improved sleep. On the other hand, improvement in sleep characteristics and symptoms may contribute to improved affect or appraisal of improvements. It also possible that CBT-I has direct biological effects on cortisol, cytokines or other biological substrates (Conley & Redeker, 2015; Irwin et al., 2015; Irwin et al., 2014) that may lead to improvements in sleep and symptoms and resulting perceptions about improvements. Given the importance of the burden of sleep-related daytime symptoms among HF patients and the need for effective symptom interventions, the mechanisms for these associations warrant further study.

Strengths of this study included the randomized controlled design with an attention-control condition, documented feasibility and acceptability of the intervention among patients with stable HF (Redeker et al., 2015), and the six month follow-up that allowed evaluation of sustained effects of CBT-I on the outcomes of interest. Limitations included the small sample and the lack of wrist actigraph measures at six month follow-up. It is also a limitation that the sample size in both groups was smaller at follow-up than at baseline. While it may appear that the attrition was higher in the attention-control group, two eligible participants from the attention-control condition obtained CBT-I and were, therefore, not eligible for followup.

We included participants who were adherent to PAP therapy for sleep apnea because of the high prevalence of this condition in HF and its frequent comorbidity with insomnia (Redeker et al., 2010). We also included those with hypnotic use because hypnotic tapering was a component of the CBT-I intervention. Although we found in our earlier study that sleep apnea did not explain insomnia (Redeker et al., 2010) in HF patients, failure to adhere to PAP may contribute to difficulty with sleep maintenance, and hypnotic use may also influence sleep. While these treatments may also influence sleep-related cognitions, the sample in this study was too small to conduct sensitivity analysis to evaluate this potential association. We also provided payments to participants that were proportional to the research requirements, but it possible that payments may contribute to perceptions about sleep or treatment. We are now conducting an NIH-funded study (Redeker et al., 2017) with a larger, full powered sample and longer term follow-up over a full year that will enable further evaluation of the effects found in this study and more fine-grained evaluation of the potential cognitive and behavioral mechanisms that may explain longterm effects of CBT-I among patients with HF.

CBT-I contributes to improvements in dysfunctional sleep-related cognitions among patients with stable HF, a group at high risk for poor sleep and excessive symptom burden. Changes in cognitions appear to be an important mechanism for improvements in insomnia severity and fatigue – both highly prevalent and disabling symptoms in this population. Continued use of CBT-I has important benefits for this vulnerable group of patients.

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Figure 1.

Figure 2.

Note. Coefficients [95% confidence interval] were estimated from 2,000 bootstrap samples

Table 1

Clinical and demographic characteristics of the total sample and the Cognitive Behavioral Therapy (CBT-I) and Attention Control Groups

Variable	Total Sample	CBT-I N=30	Attention Control N=21
Age	59.1 ± 15.1	62.0 ± 13.3	55.0 ± 17.1
Gender			
Male	25 (49.0%)	14 (46.7%)	11 (52.4%)
Female	26 (51.0%)	16 (53.3%)	10 (47.6%)
Race			
White	34 (66.7%)	19 (63.3%)	15 (71.4%)
African American	16 (31.4%)	10 (33.3%)	6 (28.6%)
More Than One Race	1 (1.9%)	1 (3.3%)	0 (0%)
Comorbidity [§] *	2.2 ± 1.5	2.5 ± 1.5	1.7 ± 1.3
NYHA [#]	2.33 ± 0.55	2.30 ± 0.60	2.37 ± 0.50
Sleep Apnea/Use CPAP	7 (13.7%)	5 (16.7%)	2 (9.5%)

[§]Charlson comorbidity Index

[#]NYHA: New York Heart Association Functional Classification

* significant difference at a 0.05 significance level.

Descriptive Statistics, Means and Standard Deviations for the Dysfunctional Beliefs and Attitudes about sleep Scale (DBAS) and Sleep Disturbance Questionnaire (SDQ) between the CBT-I and attention-control groups at time 1 and time 2.

Table 2

Variable	CBT-I			Attention Control		
	Baseline M (SD)	Time 1 M (SD)	Time 2 M (SD)	Baseline M (SD)	Time 1 M (SD)	Time 2 M (SD)
N	30	28	22	21	19	13
<i>Perpetuating Factors</i>						
Dysfunctional Beliefs and Attitudes about Sleep (DBAS)	4.64 (1.90)	3.78 (1.55)	3.47 (1.84)	4.20 (1.88)	4.61 (1.70)	4.63 (8.14)
Sleep Disturbance Questionnaire (SDQ)	2.82 (0.80)	2.33 (0.73)	2.13 (0.60)	2.44 (0.70)	2.15 (0.41)	2.23 (0.67)

Table 3

Effects of Cognitive Behavioral Therapy for Insomnia vs. Attention Control Condition on Dysfunctional Beliefs and Attitudes about sleep (DBAS) and the Sleep Disturbance Questionnaire (SDQ) after controlling for baseline age and comorbidity

	DBAS			SDQ		
	Coefficient $\pm$ StdErr	Type III F Test	(p-value)	Coefficient $\pm$ StdErr	Type III F Test	(p-value)
Age	0.017 $\pm$ 0.015	1.18	(.2813)	0.000 $\pm$ 0.005	0.00	(.9523)
Comorbidity §	-0.045 $\pm$ 0.159	0.08	(.7794)	-0.073 $\pm$ 0.056	1.65	(.2027)
Group (control)	-0.246 $\pm$ 0.543	0.21	(.6513)	-0.439 $\pm$ 0.200	4.83	(.0308)
Time	-0.600 $\pm$ 0.190	1.37	(.2444)	-0.350 $\pm$ 0.076	16.27	(.0001)
Group $\times$ Time (control)	0.842 $\pm$ 0.305	7.61	(.0072)	0.199 $\pm$ 0.124	2.56	(.1137)
<i>Estimated Slope of Time</i>		T test	(p-value)		T test	(p-value)
CBT $^{-1}$ (a)	-0.600 $\pm$ 0.190	.0023		-0.350 $\pm$ 0.076	<.0001	
Control	0.242 $\pm$ 0.239	.3133		-0.151 $\pm$ 0.098	.1277	

Coefficients and standard errors were estimated from a mixed effects model with random intercept and autoregressive correlation structure of within-subjects' changes.

§ Charlson comorbidity Index

Table 4

Correlations between Changes In DBAS, SDQ, Sleep, and Symptoms From Baseline To Post-Intervention (Time 1) And Six Month Follow-up (Time 2) for the Entire Sample

Baseline to Post Intervention (Time 1)							Change in Daytime Symptoms			
	Global Sleep Quality (PSQI) ^(a) r (p-value)	Sleep Duration – PSQI r (p-value)	Sleep Latency – Actigraphy r (p-value)	Sleep Latency-PSQI r (p-value)	Sleep Latency-Actigraphy r (p-value)	Insomnia Severity (b) r (p-value)	Fatigue ^(c) r (p-value)	Depression ^(d) r (p-value)	Anxiety r (p-value)	Sleepiness ^(e) r (p-value)
Change in DBAS	0.309 (.0412)	-0.129 (.3929)	-0.327 (.0301)	**0.369 (.0106)	0.200 (.1937)	**0.533 (.0001)	0.335 (.0213)	0.217 (.1434)	0.196 (.1855)	0.012 (.9348)
Change in SDQ	**0.381 (.0098)	** -0.524 (.0002)	-0.202 (.1839)	0.108 (.4627)	0.138 (.3655)	0.299 (.0391)	**0.398 (.0051)	**0.351 (.0143)	0.036 (.8098)	0.041 (.7822)
Baseline to Six Month Follow-up (Time 2)							Change in Daytime Symptoms			
	Global Sleep Quality (PSQI) ^(a) r (p-value)	Sleep Duration – PSQI r (p-value)	Sleep Duration – Actigraphy r (p-value)	Sleep Latency-PSQI r (p-value)	Sleep Latency-Actigraphy r (p-value)	Insomnia Severity (b) r (p-value)	Fatigue ^(c) r (p-value)	Depression ^(d) r (p-value)	Anxiety r (p-value)	Sleepiness ^(e) r (p-value)
Change in DBAS	**0.528 (.0019)	-0.085 (.6422)	N/A	**0.571 (.0005)	N/A	**0.642 (<.0001)	**0.681 (<.0001)	**0.449 (.0068)	**0.557 (.0005)	0.331 (.0598)
Change in SDQ	**0.444 (.0123)	-0.178 (.3392)	N/A	**0.381 (.0314)	N/A	0.377 (.0334)	**0.573 (.0004)	**0.538 (.0010)	**0.383 (.0253)	0.325 (.0698)

^(a) higher score = poorer sleep quality;

^(b) Insomnia Severity Index;

^(c) Global Fatigue Scale;

^(d) Centers for Epidemiological Studies of Depression Scale;

^(e) Epworth Sleepiness Scale.

** indicates significant correlations based on Benjamini-Hochberg adjustment for multiple tests at a 0.05 False Discovery Rate (FDR).