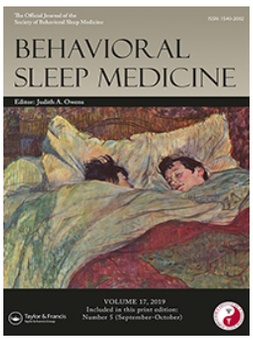

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Associations Among Trajectories of Sleep Disturbance, Depressive Symptomology and 24-Hour Urinary Cortisol in HIV+ Women Following a Stress Management Intervention

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ABSTRACT

Objective: The burden of sleep disturbance and depressive symptomology is high for persons living with HIV and particularly so for women. While cognitive behavioral stress management (CBSM) is shown to reduce symptoms of depression and 24-hr urinary free cortisol output (CORT) in HIV+ men, less is known about the effects of CBSM on mood and concomitant sleep disturbance in HIV+ women. The study aim is to model longitudinal change in sleep disturbance, depressive symptomology, and CORT for HIV+ women exposed to a 12-week CBSM intervention or control condition. *Methods*: Self-reported sleep quality and depressive symptomology, along with CORT, was collected from surveys at baseline and approximately every three months thereafter for nine months from 130 HIV+ women (M_{age} = 38.44, *SD* = 7.73). The data was used to specify a parallel process latent growth model with CORT as a time-varying covariate. *Results*: The model showed acceptable fit. There was a linear decline in sleep disturbance (β = −0.32, $p < .05$) and logarithmic decline in depressive symptomology (β = −0.33, $p < .05$) for those receiving the intervention. Decline in sleep disturbance predicted lower CORT at nine months. Furthermore, having less depressive symptoms at baseline was associated with lower initial levels of sleep disturbance and greater improvement in sleep quality over time. There was no discernible association between sleep and mood disturbance in the control group. Across groups, there was a consistent association between older age and greater sleep disturbance (r = 0.34, $p < .01$). *Conclusion*: Sleep disturbance appears to be a behavioral target for CBSM in HIV+ women although older age, preintervention levels of depressive mood, and time-varying levels of CORT output may limit improvement in sleep quality over time.

Sleep disturbance is commonly reported by persons infected with the human immunodeficiency virus (HIV). It is estimated that between 58% and 74% of persons living with HIV (PLWH) have difficulty initiating or maintaining sleep or suffer from early morning awakening (Reid & Dwyer, 2005; Wu, Wu, Lu, Guo, & Li, 2015). Several factors are attributed to the severity of sleep disturbance in PLWH such as HIV-disease duration, symptom burden, living condition, and psychological distress (Nokes & Kendrew, 2001; Robbins, Phillips, Dudgeon, & Hand, 2004).

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These studies collectively implicate elevated levels of depressive symptomology as a strong predictor of sleep disturbance in PLWH. It is also clear that the burden of self-reported sleep disturbance is higher in women (69%) than in men (48%) (Wu et al., 2015). Although sleep disturbance is identified as a key area of concern for female PLWH, little is understood regarding the interrelationships among sleep, depression, and hypothalamic-pituitary-adrenal (HPA) activity and how these associations may change over time and as a function of chronic disease progression (Field et al., 2007; Gur, Cevik, Nas, Colpan, & Sarac, 2004; Gur, Cevik, Sarac, Colpan, & Em, 2004; Payne, Held, Thorpe, & Shaw, 2008; Payne, Piper, Rabinowitz, & Zimmerman, 2006; Steiger, 2003; Stetler & Miller, 2005). Despite the heightened risk for depression and sleep disturbance in women and PLWH, very little is known regarding the natural trajectory of this symptom burden in HIV+ women. Thus, we sought to investigate the longitudinal associations between self-reported sleep quality, depressive symptoms, and cortisol output among women living with HIV who were assigned to a stress management intervention or wait-list condition.

Women in the general population are more likely to report symptoms of insomnia and experience daytime consequences of poor sleep; they are twice as likely as men for having an insomnia diagnosis (Marion et al., 2009). Women are also more likely to report depression that is closely related to sleep disturbance (Ohayon, 2002). Moreover, the magnitude of sleep disturbance appears to be compounded by HIV disease severity, as a number of women report strong associations between CD4 T-cell count and self-reported sleep quality (Junqueira, Bellucci, Rossini, & Reimão, 2008; Lui-Filho et al., 2013; Marion et al., 2009; Seay et al., 2013). One of the largest studies comparing sleep disturbance in women as a function of HIV status found PLWH were 26% more likely to endorse symptoms of insomnia than their HIV-negative counterparts (Jean-Louis et al., 2012). Although this study found sleep disturbance did not vary as a function of disease-related variables, (i.e., viral load, CD4 T-cell count, and disease duration), women of postmenopausal age were at increased risk for insomnia.

Major depressive episodes appear to be heavily concomitant with sleep disturbance in adults (Ford & Kamerow, 1989; Ohayon, 2002). Among PLWH, the incidence of major depression has been estimated as nearly twice that of healthy controls (Ciesla & Roberts, 2001). This elevated risk for depression and associated symptomology is attributed to HIV disease severity, substance abuse, female gender, and various psychosocial factors such as stigma and isolation (Asch et al., 2003; Ciesla & Roberts, 2001; Rabkin, 2008). A number of cross-sectional studies report elevated depressive symptomology in individuals reporting poor sleep quality (Allavena et al., 2016; Crum-Cianflone et al., 2012; Lee et al., 2012; Nokes & Kendrew, 2001; Phillips et al., 2005; Rubinstein & Selwyn, 1998), indexed by a global score of > 5 on the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Among HIV+ women, the associations are more tenuous, as poor sleep quality was linked with depressive symptomology in one study (Marion et al., 2009), yet not in two others (Junqueira et al., 2008; Lui-Filho et al., 2013).

It is widely suggested that dysfunction of HPA axis resulting in elevated levels of glucocorticoids is concomitant with symptoms of depression and insomnia (Arborelius, Owens, Plotsky, & Nemeroff, 1999; Buckley & Schatzberg, 2005). The direction of these associations is unclear, as HPA axis overactivity in response to stress contributes to the pathophysiology of depression (Pittenger & Duman, 2008) as well as sleep disturbance (Meerlo, Sgoifo, & Suchecki, 2008). On the other hand, sleep disturbance is linked to elevated urinary free cortisol (UFC; Vgontzas et al., 2001, 1998) and plasma cortisol output (Rodenbeck & Hajak, 2001; Rodenbeck, Huether, Rüther, & Hajak, 2002), independent of depression. Although there is evidence of elevated UFC in PLWH reporting higher levels of depression (Enwonwu, Meeks, & Sawiris, 1996; Leserman, 2003), the relationship of depression to sleep disturbance over time, amongst HIV+ women, is unclear.

If the underlying neurobiology of sleep disturbance, depressive symptomology, and UFC output are interrelated and the latter two are shown to be ameliorated by cognitive-behavioral stress management (CBSM) intervention in HIV+ men (Antoni et al., 2000), then it is possible these effects might extrapolate to sleep problems in HIV+ women. Allied work in women with early stage

breast cancer found that the skills provided by CBSM to more effectively manage stress and utilize social resources reduced negative affect (Antoni et al., 2006), PM serum cortisol levels (Antoni et al., 2009) and self-reported sleep disturbance (Vargas et al., 2014). We hypothesize that these documented effects for a 10-week CBSM on depression and UFC output may also translate to improvement of sleep quality in female PLWH.

Study objectives

The current study is a secondary analysis of previously conducted randomized controlled trials (RCT) of CBSM in HIV+ women (Lechner et al., 2003) that utilize a latent growth curve analysis (Llabre, Spitzer, Siegel, Saab, & Schneiderman, 2004) to test three hypotheses. First, it was hypothesized that HIV+ women receiving CBSM would show reduced sleep disturbance, 24-hr UFC and depressive mood over nine months compared to women randomized to a one-day behavioral stress management seminar control condition, after controlling for age and disease-related characteristics. Second, we hypothesized that greater levels of 24-hr UFC would relate to greater sleep disturbance and depressive mood at each time point. Third, we hypothesized that changes in sleep disturbance would parallel changes in 24-hr UFC and depressive mood over nine months.

Methods

Participants were HIV+ women recruited between 1993 and 1997, through flyers and direct referrals from physicians and service organizations, to take part in an RCT where they would receive either a 10-week CBSM intervention study or were a control condition (Antoni et al., 2000, 2005; Cruess et al., 2000, 1999, 2000; Lutgendorf et al., 1997). Upon arrival for assessment, referrals received an informed consent and briefing on the study. After signing the consent form they underwent a psychiatric interview and a brief cognitive functioning screen by a trained technician. Because participants with severe mood disturbances or other forms of major psychopathology may show significantly different hormonal profiles and have difficulties in CBSM groups, we screened out individuals with scores on the Hamilton Rating Scale for Depression indicative of severe mood disturbance (> 15). The Mini Mental Status Exam was used to screen for the presence of gross neurocognitive dysfunction (< 25). Interviews were administered via trained examiners, and questionnaires were self-administered.

Participants were excluded if they had a prior history of AIDS diagnosis (Castro et al., 1993) or T-helper cell count less than 200 cells/mm3, were prescribed medications with immunomodulatory effects (e.g., cytotoxic chemotherapy, corticosteroids, or interferons), had a history of chemotherapy or whole-body radiation treatment for non–AIDS-related cancer, had a history of chronic immune illness, Type 1 diabetes mellitus, chronic hepatitis, asthma, or chronic fatigue syndrome, or a history of smoking > 50 packs of cigarettes per year. Other exclusion criteria included blood transfusions prior to 1985, antibiotic use for acute infection contracted within the past two weeks, changes in antiretroviral medication regimen over the past two months, hospitalization for surgery within the past three months, and intravenous drug use within the previous six months. In addition, participants were screened out at the initial appointment based upon the presence of severe cognitive impairment based upon scores of the HIV Dementia Scale below 10 out of 16 points (Power, Selnes, Grim, & McArthur, 1995), illiteracy at the sixth-grade level, current psychosis, drug or alcohol dependence, active suicidality, and major psychiatric disorders such as bipolar affective disorder, anxiety disorder, borderline personality disorder, and panic attacks using the Structured Clinical Interview for DSM-IV (First, Spitzer, Gibbon, & Williams, 1997).

Sixty-two women were enrolled in the wait-list condition and a total of 82 in the 10-week CBSM condition. A total of 14 women completed baseline but did not return after time point 1 and were subsequently removed from the study, leaving a total of 55 women from the control condition and 75 from the CBSM intervention. The patients who were enrolled in the control condition completed

the same assessments as those in the 10-week CBSM condition. In contrast to the 10-week wait-list period, individuals in the control condition were offered a 1-day didactic and experiential stress management workshop that provided a summary of the concepts presented during the 10-week CBSM arm. During the initial visit, all participants completed an informed consent, provided morning peripheral venous blood samples, and received a physical examination from a physician. Participants were assessed at baseline (T1), two weeks post-CBSM and three months after baseline (T2), six months after baseline (T3), and at nine months after baseline (T4).

Intervention

Participants in the CBSM condition attended 10 small weekly 135-min sessions (45-min relaxation component and 90-min stress management component) and were instructed to practice relaxation exercises twice daily between sessions. "The focus of the CBSM is on increasing awareness of the physiological effects of stress, cognitive-behavioral theory of stress and emotions, identifying cognitive distortions and automatic thoughts, cognitive restructuring, coping skills training, assertiveness training, anger management, and strategies for identifying and utilizing social supports." The intervention was led by two group facilitators (postdoctoral fellows and advanced doctoral graduate students) according to a detailed training manual and were supervised weekly by a licensed clinical psychologist and a board-certified psychiatrist. Following a 10-week waiting period, participants in the control condition completed identical assessments to those in the CBSM condition and were offered a 1-day stress management workshop summarizing several of the concepts presented in the CBSM sessions 2 weeks following 9-months after baseline.

Measures

Depression

The depression–dejection subscale of the Profile of Mood States (POMS) (McNair, 1971) was used to measure depressed mood over the 7 days prior to the study assessment. The POMS is a 65-item scale that assesses six different mood states using various adjectives, which participants rated on a 5-point Likert scale, with 0 = not at all and 4 = extremely. The POMS has good internal reliability (r >.90) and excellent construct validity (McNair, 1971). A number of HIV studies have used the POMS to assess mood status (Antoni et al., 1991; Carrico et al., 2006, 2005; Cruess et al., 2002; Olatunji, Mimiaga, & Cleirigh, 2006; Schneiderman, 1999). Depression subscale scores showed good reliability across the four time points (α = .91) in the present sample and were entered into the model to represent depressive mood.

Urinary free cortisol

We used 24-hr urinary free cortisol (CORT) in order to provide an index of free cortisol proportional to biologically active levels available in the circulation (Kuhn, 1989). Cortisol circulates in the blood in both free and bound forms; we assayed the free form, which is shown to have a biological half-life of approximately 80 min. Participants were given a urine collection container that contained 1 g of the preservative sodium metabisulfite for collection of 24-hr samples at study entry, after the intervention, and six months postintervention. They were provided with verbal and written instructions informing them as to how to collect their urine and abstain from caffeine, alcohol, antihistamines, and nicotine beginning the night before collection and continuing until completion. Specifically, participants were told to begin collecting on the second urination of the first day, to continue through the first urination of the second day, and to keep their samples refrigerated until they were dropped off at the assessment facility. All the samples received from the participants were measured for volume, and then two aliquots of 10 ml each were frozen at −70°C until being assayed.

The choice to collect and assay 24-hr urinary free cortisol was made because of several noted advantages. For example, plasma-free cortisol is shown to be technically difficult to measure due to

the need to separately determine bound and unbound fractions; in contrast, 24-hr UFC reflects ony the unbound or "free" fraction. Second, while plasma cortisol reflects the release of cortisol over the past 20–30 min, our UFC measure captures cumulative cortisol output over a 24-hr period. Although UFC is not able to account for the fluctuations in cortisol associated with circadian rhythmicity (which would require separate samples evaluated at intervals across the diurnal period), UFC was chosen as a measure of general adrenal output.

Immune measures

Peripheral blood samples were collected to determine CD3+CD4+ cell counts at each time point. Blood samples were collected between 8 A.M. and 12 P.M. using sterile evacuated tubes containing sodium heparin (Vacutainer Cat #6489, Becton-Dickinson, Rutherford, NJ). A single laser flow cytometer (EPICS C, Coulter Instruments Laboratories, Hialeah, FL) was used with a whole blood, two-color immunofluorescence analysis, as described elsewhere (Fletcher, Baron, Ashman, Fischl, & Klimas, 1986). The percentage of cells positively stained for CD3 and CD4 were converted to an absolute count by multiplying by the lymphocyte count obtained from a MAxM hematology counter (Coulter Instruments Laboratories, Hialeah, FL). The primary immunologic outcome variable used in this study was the CD3+CD4+ cell count at the nine-month follow-up period.

Sleep quality

The 18-item Pittsburgh Sleep Quality Index (PSQI) was used to measure multiple dimensions of sleep quality (Buysse et al., 1989). The PSQI global score produces scores ranging from 0 to 21, with higher scores indicating poorer overall sleep quality. The PSQI global score is comprised of seven component scores that can be analyzed separately to examine subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. The PSQI global score was calculated using the algorithm specified in Buysse et al. (1989). Although the PSQI has been validated in chronic disease populations including chronic pain, breast cancer, and depression, there have been no concerted efforts to norm the scale in HIV populations (Buysse et al., 1989). However, a recent analysis purported its usefulness compared to nonstandardized or informal assessments of sleep quality in HIV patients. The meta-analysis reported that responses on the PSQI did not vary as a function of gender and age (i.e., < 40 and > 40 Wu et al., 2015).

HIV-1 viral load

Serum viral load (VL) was measured by determining the number of HIV-1 virions per milliliter (ml) of peripheral blood plasma using the Cobas Amplicor HIV-1 Monitor RT/PCR assay (version 1.5, Roche Molecular Systems, Branchburg, NJ). Lower limit of sensitivity of this assay was 400 copies of HIV-1 RNA/ml of plasma.

Analysis

A parallel-process latent growth model (LGM) with time-varying covariates was used to establish growth trajectories (i.e., increases or decreases across time) by specifying the mean for global sleep disturbance and depressed mood at each time point. The latent variable for the initial observation point (intercept) and linear change in this factor over time (slope) were modeled from observations at baseline (T1), three months after baseline (T2), six months after baseline (T3), and nine months after baseline (T4). Beyond comparing the intercept and slope between intervention conditions, the time-varying relationship of the slope and intercept with 24-hr UFC was examined in a second model and a third including time-invariant measures of baseline age, HIV-disease duration, CD4 count, and viral load, given previously reported associations with postmenopausal status and HIV disease status on sleep disturbance in female PLWH (Junqueira et al., 2008; Lui-Filho et al., 2013; Seay et al., 2013).

For all models, the full maximum likelihood estimation procedure was utilized in Mplus version 7.11 (Muthén & Muthén, 1998) to model all data available from each participant, assuming that data are missing at random (McArdle, 1994). Model assessment included chi-square statistics, comparative fit indices (CFI), and root means of the standard estimate (RMSEA). The criteria used to determine acceptable model fit included CFI ≥ .90, SRMR ≤ 0.10, and RMSEA < .08 (Hu & Bentler, 1999). Furthermore, the goodness-of-fit criterion was designated as failure to reject the null hypothesis in the model $\chi 2$ fit test. Within each model, the parameter estimates were evaluated by their critical ratio. Due to limitations in sample size, the variance of sleep disturbance and depressed mood were constrained to zero at each time point.

Results

Table 1 shows the demographic and HIV disease-related variables for each group at baseline. There were no significant differences in age or ethnic distribution, CD3+CD4+, CD3+CD8 + T-cell count or HIV viral load; nor were there any group differences in mean cortisol, sleep disturbance, or depressed mood. Trajectories for sleep disturbance, cortisol, and depression over nine months, for both the CBSM and wait-list group, can be found in Figure 1. Women who completed the baseline assessment and did not return for any of the other three time points were excluded from the analysis. Attrition was compared as a function of condition for each observed time point of sleep disturbance and depressed mood. Attrition over the four time points (nine months) was 30% for the wait-list group and 38% for the CBSM group and did not vary as a function of CBSM group (see Table 3).

Parallel latent growth analysis

Table 2 provides parameter estimates and confidence intervals for the parallel latent growth model of sleep disturbance and depressed mood for women exposed to the control (see Figure 2) and CBSM condition (see Figure 3). The model produced a nonsignificant chi-square test statistic $\chi 2 = 55.75$, $p = 0.11$, and fit indices representing an acceptable model fit, CFI = 0.97, RMSEA = 0.06, SRMR = 0.11 (Cruess et al., 1999). Only the intercept for sleep disturbance and depressed mood was significant in the control group, suggesting the absence of linear change in these variables over time. In the CBSM condition, both intercept and slope were significant for sleep disturbance and depressed mood, suggesting a decline in both parameters over time.

The intercorrelations for slope and intercept were not significant in the control condition. Women assigned to the CBSM condition with greater initial levels of sleep disturbance reported greater depressed mood and a slower decline in sleep disturbance over time. Those reporting the greatest levels of baseline depressed mood showed the slowest decline in depressed mood over time. There was no significant relationship between initial levels of depressed mood and change in sleep disturbance. There was a marginally significant ($p = .09$) association between the slope of sleep disturbance and depressed mood, such that greater decreases in sleep disturbance over time related to greater reductions in depressed mood within the CBSM group.

Time-variant covariates

The inclusion of time-varying measures of 24-hr UFC produced a significant chi-square test statistic $\chi 2 = 136.08$, $p < .001$(Cruess et al., 1999), and minimally acceptable fit indices, CFI = 0.90, RMSEA = 0.09, SRMR = 0.16. In the control group, higher initial sleep disturbance related to elevated cortisol at T3. In the CBSM group, greater baseline levels of depressed mood related to higher UFC at T2. Greater reduction in sleep over time was related to lower UFC at the end of study within the CBSM condition.

Table 1. Demographic and disease-related characteristics of the cohort.

Characteristics	Wait-list Mean ± *SD*	CBSM Mean ± *SD*	*F* value
Age (years)	39.20 ± 8.13	37.88 ± 7.43	0.93
Ethnicity (%)			
Black, non-Hispanic	71.0	69.5	
Hispanic	11.3	18.3	
White, non-Hispanic	3.2	2.4	
Other	3.2	2.4	
Education (%)			−0.30
Less than 12 years	38.7	44.7	
H.S. graduate	24.2	27.6	
Some college/trade school	17.7	18.4	
College graduate & beyond	9.7	9.2	
Household income ($/year; %)			−0.12
Less than 10 k	32.3	32.9	
10,001–20,000 k	35.5	39.0	
20,001–30,000 k	19.4	14.6	
30,001 and over	3.2	3.4	
Disease variables			
Time since HIV diagnosis (months)	84.4 ± 61.5	84.4 ± 61.5	1.30
Current CD4+ count (cells/μl)	470.06 ± 343.69	500.20 ± 288.55	0.28
Current CD8+ count (cells/μl)	891.15 ± 544.32	935.42 ± 471.82	0.24
HIV-1 viral load current (log_{10})	1.92 ± 1.78	2.39 ± 1.52	2.52
Antiretroviral medication (%)	54.6	58.9	0.01
Protease inhibitor	5.4	10.5	
Nucleoside reverse transcriptase inhibitors	41.1	42.1	
Nonnucleoside reverse transcriptase inhibitors	7.1	5.3	
T1 anti-HIV medication adherence (4-day)	89.4	95.1	1.45
T2 anti-HIV medication adherence (4-day)	90.5	91.5	0.04
T3 anti-HIV medication adherence (4-day)	90.0	93.7	0.51
T4 anti-HIV medication adherence (4-day)	97.1	90.1	1.32
Neuroendocrine			
T1 24-hr urinary cortisol output	4.32 ± 3.60	4.05 ± 3.27	0.17
T2 24-hr urinary cortisol output	4.12 ± 3.31	4.00 ± 3.21	0.03
T3 24-hr urinary cortisol output	5.78 ± 10.3	4.09 ± 4.32	0.22
T4 24-hr urinary cortisol output	3.93 ± 3.54	5.28 ± 5.10	1.42
Sleep disturbance			
T1 global sleep disturbance	5.88 ± 3.68	6.11 ± 3.82	0.11
T2 global sleep disturbance	5.83 ± 4.06	5.14 ± 3.66	0.88
T3 global sleep disturbance	6.05 ± 4.37	5.06 ± 3.55	1.51
T4 global sleep disturbance	5.69 ± 4.48	4.91 ± 3.80	0.73
Depression			
T1 Depressive symptomology	4.00 ± 6.36	4.77 ± 8.91	0.30
T2 Depressive symptomology	2.18 ± 3.87	3.23 ± 7.75	0.77
T3 Depressive symptomology	2.60 ± 3.10	2.97 ± 6.15	0.14
T4 Depressive symptomology	2.89 ± 6.02	3.30 ± 6.15	0.09

Time-invariant covariates

Additional analyses were conducted to determine whether time-invariant disease and sociodemographic covariates relate to intercept or slope for sleep and depressed mood. In order to estimate this model, time-variant measures of UFC as well as the time-invariant measures were specified to freely correlate with the latent factors for sleep and depression. After specifying these paths with age, disease duration, baseline CD4 count, and viral load, the model demonstrated poor fit $\chi 2$ (164) = 333.24, $p < .001$, CFI = 0.75, RMSEA = .125, SRMR = 0.16. There were, however, some notable paths that were significant. In the control condition, initial levels of sleep disturbance correlated with older age ($\beta = 0.29$, $p < .05$) and lower viral load ($\beta = -0.25$, $p < .05$). There was a similar finding between age and sleep disturbance in the CBSM group ($\beta = 0.37$, $p < .001$); however, initial sleep disturbance was also associated with a shorter disease duration ($\beta = -0.30$, $p < .01$). There was a positive association between

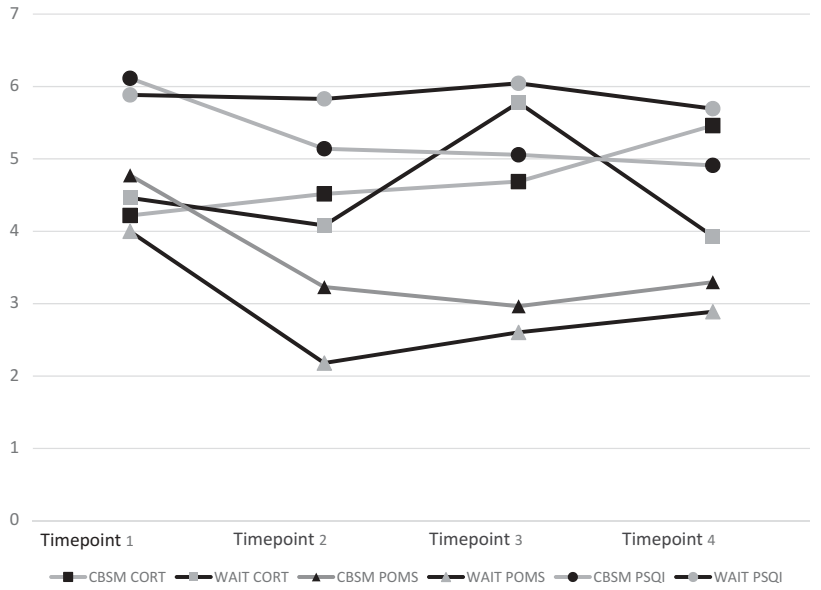

Figure 1. Mean 24-hr urinary cortisol output (CORT-square), depressive symptomology (POMS-triangle, and sleep disturbance (PSQI-circle) for CBSM and wait-list group at baseline, three months, six months, and nine months.

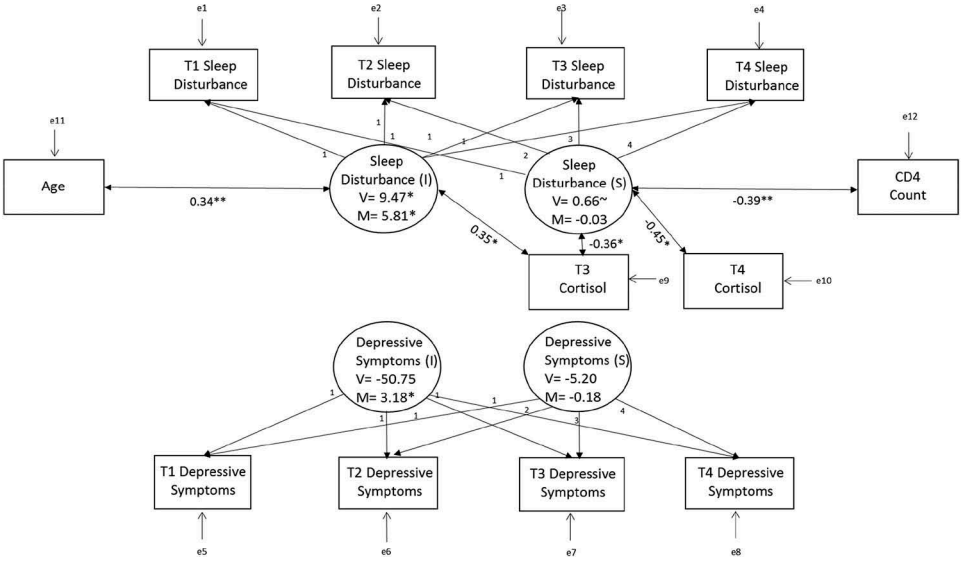

Figure 2. Parallel process latent growth model for women in the control condition (n = 55) with time-variant and time-invariant variables.

the slope of sleep disturbance and both disease duration (β = 0.34, p < .01) and baseline CD3+CD4+ count (β = 0.21, p < .05), suggesting women with longer disease duration and higher T-helper cell count showed a greater linear decrease in sleep disturbance following CBSM. Higher initial levels of depressed mood were associated with lower CD3+CD4+ cell count (β = −0.16, p < .05) and shorter disease duration (β = −0.19, p < .05). Greater initial levels of depression were also related to older age (β = 0.23, p < .01). Conversely, older age was associated with slower decline in depressed mood (β = −0.16, p < .05).

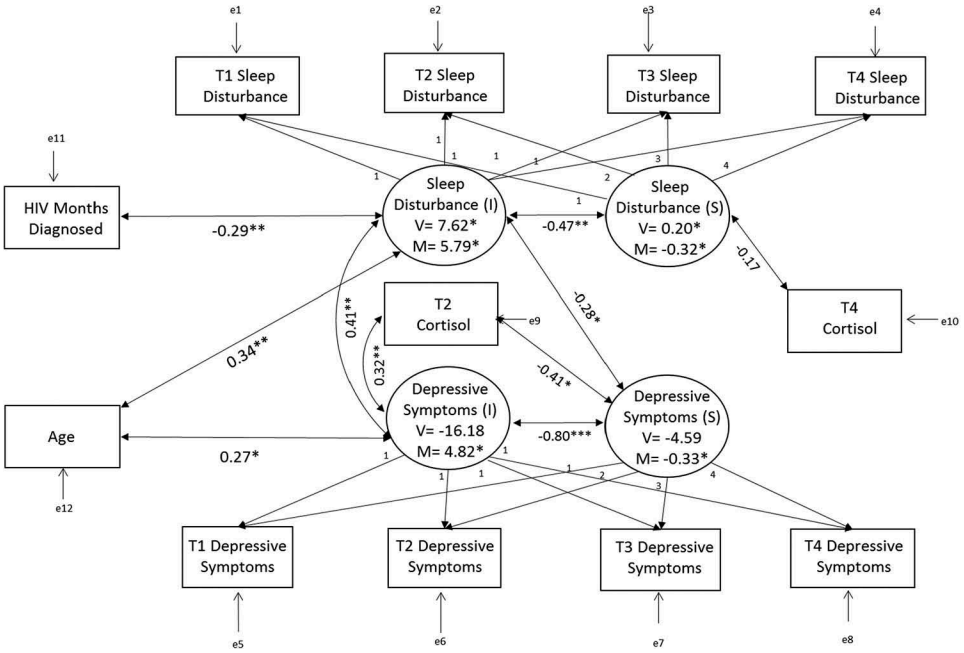

Figure 3. Parallel process latent growth model for women in the CBSM condition (n = 75) with time-variant and time-invariant variables.

Discussion

This study examined, for the first time to our knowledge, whether the trajectories of sleep disturbance and depressed mood are affected by exposure to a 10-week CBSM intervention. The central finding in this study was a linear decline in global sleep disturbance (i.e., increase in sleep quality) and a parallel decline in depressed symptoms among HIV+ women assigned to the CBSM condition versus controls. However, 24-hr urinary free cortisol output was not systematically associated with mood and sleep disturbance in this study. Since the women assigned to the two different study conditions were nearly identical in terms of sociodemographic background, HIV-disease severity, HPA-axis output, and initial levels of both sleep disturbance and depressed mood, it is evident that exposure to a 10-week CBSM may result in positive change in the trajectory of sleep quality and depressed mood for female PLWH.

There were no discernible patterns of coincidence between the parameters of sleep disturbance and depressed mood within the wait-list condition. Women randomized to the CBSM condition that reported higher initial levels of depression showed a slower decline in the severity of depressive mood over time. Higher depression at baseline also corresponded with poorer sleep quality in the CBSM condition. Moreover, greater initial levels of depressed mood corresponded negatively with improvements sleep quality. These results provide some evidence of the efficacy of CBSM on sleep quality as a secondary outcome in female PLWH, albeit this process may be somewhat dependent upon parallel reductions in depressed mood. Longitudinal research has established a temporal link between sleep disturbance and the development of depression in healthy adults (Baglioni et al., 2011; Breslau, Roth, Rosenthal, & Andreski, 1996; Ford & Kamerow, 1989; Riemann & Voderholzer, 2003). Exactly what aspect of sleep disturbance relates most strongly to depressed mood in this study is unclear. Various indices of sleep disturbance, including sleep apnea symptoms in the absence of insomnia (Peppard, Szklo-Coxe, Hla, & Young, 2006), predict higher levels of depression over time in clinically depressed persons (Arfken et al., 2014).

Table 2. Model parameter estimates for wait-list and CBSM groups.

	Wait-list				CBSM			
Parameter	Estimate	Low	High	*p*	Estimate	Low	High	*p*
Mean								
Sleep disturbance intercept	5.81	4.93	6.70	<.001	5.79	5.01	6.57	< .001
Sleep disturbance slope	−0.03	−0.34	0.29	n.s.	−0.32	−0.58	−0.05	< 0.05
Depression intercept	3.18	2.00	4.35	<.001	4.82	2.58	7.06	< .001
Depression slope	−0.18	−0.46	0.09	n.s.	−0.33	−0.74	0.08	< .05
Cortisol 1	4.44	3.50	5.39	< .001	4.25	3.44	5.05	< .001
Cortisol 2	4.19	3.22	5.16	< .001	4.85	3.54	6.17	< .001
Cortisol 3	5.36	3.02	7.70	< .001	4.85	3.63	6.07	< .001
Cortisol 4	4.12	2.82	5.42	< .001	5.21	3.82	6.60	< .001
Variance								
Sleep disturbance intercept	9.47	6.11	12.83	< .001	11.17	7.62	14.71	< .001
Sleep disturbance slope	0.66	−0.05	1.38	0.07	0.91	0.20	1.61	0.01
Depression intercept	−50.75	−123.77	22.26	n.s.	87.30	−16.18	190.78	0.10
Depression Slope	−5.20	−11.78	1.37	n.s.	2.17	−4.59	8.92	n.s.
Cortisol 1	11.92	5.18	18.66	.001	12.41	4.54	20.27	.002
Cortisol 2	10.32	6.48	14.16	< .001	20.06	2.53	37.59	0.03
Cortisol 3	107.27	−73.67	288.2	n.s.	20.70	10.11	31.29	< .001
Cortisol 4	12.09	1.87	22.31	0.02	24.33	5.68	42.97	0.01
Correlations								
SP with IP	−0.09	0.29	−0.33	n.s.	−0.47	−3.00	0.05	0.004
SD with SP	< .01	0.34	−0.85	n.s.	0.11	−0.17	0.47	0.09
SD with IP	< .01	0.30	0.45	n.s.	−0.28	−3.17	0.38	< 0.05
SD with ID	< .01	1.99	0.41	n.s.	−0.80	−37.04	14.96	< .001
ID with IP	< .01	1.85	0.82	n.s.	0.41	3.68	22.19	< 0.01
IP with CORT T1	−0.17	−5.11	1.41	n.s.	−0.07	−3.30	1.77	n.s.
IP with CORT T2	−0.15	−4.34	1.31	n.s.	0.10	−2.04	4.92	n.s.
IP with CORT T3	0.35	−8.18	30.21	< 0.05	0.20	−1.59	7.57	n.s.
IP with CORT T4	−0.04	−3.50	2.75	n.s.	−0.15	−8.40	3.31	n.s.
SP with CORT T1	0.11	−0.23	0.84	n.s.	0.03	−1.04	1.27	n.s.
SP with CORT T2	0.16	−0.96	1.81	n.s.	−0.04	−1.40	1.03	n.s.
SP with CORT T3	−0.36	−8.16	2.03	0.06	−0.22	−2.52	0.63	n.s.
SP with CORT T4	−0.45	−2.74	0.22	0.04	0.14	−0.92	2.22	n.s.
ID with CORT T1	< .01	−1.48	6.16	n.s.	0.02	−4.71	6.01	n.s.
ID with CORT T2	< .01	−2.57	3.86	n.s.	0.32	−4.13	30.94	0.01
ID with CORT T3	< .01	−14.01	10.84	n.s.	0.01	−3.52	12.11	n.s.
ID with CORT T4	< .01	−1.98	11.75	n.s.	−0.07	−22.66	16.00	n.s.
SD with CORT T1	< .01	−1.07	0.30	n.s.	−0.15	−1.68	0.16	n.s.
SD with CORT T2	< .01	−0.91	0.81	n.s.	−0.41	−6.36	1.00	0.08
SD with CORT T3	< .01	−8.16	2.03	n.s.	−0.22	−2.74	−0.25	n.s.
SD with CORT T4	< .01	−2.74	0.22	n.s.	0.18	−1.94	4.62	n.s.
24-hr urinary cortisol output (CORT)								

Levels of UFC over the nine-month study period were unrelated to the slope and intercept of sleep disturbance and depressed mood in the CBSM group; however, greater initial levels of depressed symptoms were related to higher UFC output at the subsequent time point. HPA-axis dysfunction is central to the etiology of depression and insomnia (Meerlo et al., 2008; Pittenger & Duman, 2008). Moreover, changes in UFC are found to covary with the recurrence of major depression and depressed mood over time (Rao et al., 1996). However, it should be noted that those HIV studies showing CBSM-related decreases in mood and cortisol level exclusively featured salivary cortisol measurements (Antoni et al., 2000; Cruess, Antoni, Kumar, & Schneiderman, 2000). The 24-hr urinary cortisol measure gathered for this study was intended to provide an index of the total amount of cortisol released by the adrenals over a complete circadian cycle. In contrast, the unbound cortisol assayed from diurnal measurements of saliva more consistently reflects the relationship between sleep disturbance and depressed mood HPA-axis activity (Vgontzas et al., 2001). Thus, it is possible that our urinary cortisol measures might be obscuring subtler effects among mood, sleep disturbance, and adrenal output.

Table 3. Chi-square tests for CBSM group differences in attrition.

Parameter estimates	Time point 1–2		Time point 2–3		Time point 3–4	
	$\chi^{2(df,n)}$	Value (sig)	$\chi^{2(df,n)}$	Value (sig)	$\chi^{2(df,n)}$	Value (sig)
Sleep quality	1(Asch et al., 2003)	0.01	1(Rodenbeck et al., 2002)	1.29	1(Power et al., 1995)	0.01
Depressive symptoms	1(Jean-Louis et al., 2012)	0.46	1(Rubinstein & Selwyn, 1998)	1.81	1(Antoni et al., 2005)	0.82

Limitations

First, it is acknowledged the present study is a post-hoc secondary analysis of a previously conducted trial that was not designed to examine sleep-related changes as a primary outcome. The fact that all of the study participants were enrolled in a behavioral intervention trial also limits our ability to generalize these findings to changes in sleep and depressed mood that could be expected in the larger population of HIV-infected women. Second, although the PSQI shows high internal consistency in chronic disease populations and high criterion validity, indexed by strong correlations with sleep-related items and subscales on other measures, the self-report index shows poor concurrent validity, as has been evidenced by comparing scores to wrist actigraphy and polysomnographic measures of sleep (Buysse et al., 1989; Carpenter & Andrykowski, 1998; Grandner, Kripke, Yoon, & Youngstedt, 2006). This suggests that the PSQI reflects a subjective measure of sleep quality, but falls short of indexing actual disturbance of sleep. The inherent limitations of self-report sleep quality, albeit through a well-normed and reliable psychometric instrument (Buysse et al., 1989), apply to the current findings and in the future might be bolstered by objective measures such as actigraphy, slow-wave EEG, and obstructed breathing during sleep. Nevertheless, there is compelling evidence for the relationship between self-reported sleep quality and depressed mood. Third, construct validity is often an issue as it pertains to sleep report in PLWH because greater report of depression, anxiety, and fatigue may inflate self-report of sleep disturbances in this population. Fourth, while this study provided some evidence of parallel changes in depression with improvement of sleep quality after this intervention, other behavioral variables (i.e., variable sleep and wake times, television viewing, and consumption of caffeine) have also been implicated in the etiology of poor sleep hygiene in PLWH (Downing et al., 2016; Nunnari et al., 2016). Furthermore, chronic antidepressant therapy may develop a serotonin syndrome that can alter the impact of sleep disturbance on depressed mood (Adrien, 2002; DeSilva, Le Flore, Marston, & Rimland, 2001). Fifth, although we uncovered a pattern of interrelationships between time-dependent and time-independent measures of sleep quality and depressed mood, the model did not explicitly test causal effects due to limitations in sample size. Small sample size and group imbalances due to attrition are a major limitation for the study, as attrition reduces the statistical power of the analyses. However, we incorporated the maximum likelihood estimation feature from Mplus that has been shown to provide robust estimates for high attrition-rate data. Sixth, the 24-hr urinary cortisol measure gathered for this study was intended to provide an index of the total amount of cortisol released by the adrenals over a complete circadian cycle, but it lacks sensitivity to diurnal changes in reflecting adrenocortical release that is more accurately measured in the saliva. It has been well demonstrated that hypercortisolemia and peripheral resistance to glucocorticoids are characterized by abnormal glucocorticoid receptors on lymphocytes in HIV patients (Suls, Wan, & Costa, 1995). Based on this association, we included CD4 T-lymphocyte count as a time-invariant covariate of interest.

It should also be noted that because the PSQI indexes sleep quality over the past month and the POMS provides a subjective report of depressive symptoms over the last week, it would appear that there may be some temporal precedence for sleep disturbance on depressive symptomology. There may be a greater likelihood that self-reported depressive symptomology might reflect cumulative

change in quality of sleep than vice versa. It is for these reasons that we modeled the relationship between intercept and slope of sleep disturbance and depressive symptomology as bidirectional associations rather than paths that are indicative of temporal precedence.

Conclusion

Female gender is systematically shown to be a risk factor for insomnia in PLWH (Wu et al., 2015). The established coincidence of change in major depression and insomnia in this population is of importance due to the negative cardiovascular, metabolic, and immune outcomes with which they are linked (Kiecolt-Glaser & Glaser, 2002; Motivala, 2011; Taylor, Lichstein, & Durrence, 2003). Here, we provide preliminary evidence that female PLWH assigned to a group-based stress management intervention report reduced symptoms of depression and insomnia compared to those in a wait-list condition. Future work should explore the interaction of behavioral, neuroendocrine, immune, and viral processes that could influence the seemingly parallel trajectory of mood and sleep disturbance in PLWH that was observed in this study. Although the clinical ramifications of an HIV diagnosis have changed since the time this data was collected, extrapolation of these findings about PLWH in the 21st century is suitable due to the universality of the CBSM approach, wherein the focus is increasing awareness of the physiological effects of stress, applying cognitive-behavioral theory of stress and emotions to identify cognitive distortions and automatic thoughts, and then use cognitive restructuring, coping skills training, assertiveness training, anger management, and strategies for identifying and utilizing social supports to better manage stressors.

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