

Effect of depression treatment on subjective sleep components among primary care patients: Data from the eIMPACT trial

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ABSTRACT

Objective: Sleep disturbance is a multifaceted symptom of depression that disproportionately impacts marginalized groups. Depression treatment improves sleep disturbance in some individuals; however, the components of sleep disturbance improved remain unidentified. This secondary analysis of the eIMPACT randomized controlled trial examines effects of depression treatment on subjective sleep disturbance components.

Methods: 216 primary care patients with depression from a safety net healthcare system were randomized to 12-months of modernized collaborative care (internet cognitive-behavioral therapy [CBT], telephonic CBT, and/or antidepressants; $n = 107$) or usual primary care for depression (primary care providers supported by embedded behavioral health clinicians and psychiatrists; $n = 109$). Subjective sleep disturbance components were assessed by the Pittsburgh Sleep Quality Index (PSQI).

Results: Mixed-effect models examined intervention effects on changes in subjective sleep disturbance components. The intervention improved PSQI global scores, sleep onset latency, subjective sleep quality, and daytime dysfunction across 24-months, and these effects diminished after treatment termination. At post-treatment, intervention participants had greater improvements in PSQI global scores ($p < 0.001$, $d = -0.62$), sleep onset latency ($p < 0.01$, $d = -0.43$), daytime dysfunction ($p < 0.01$, $d = -0.35$), and sleep disturbances ($p = 0.01$, $d = -0.26$) compared to usual care, but no differences in subjective sleep quality, total sleep time, sleep efficiency, or sleep medication use. Intervention effects were not moderated by race, education, or income. Pre- to post-treatment improvements in depressive symptoms were associated with improvements in some sleep disturbance components.

Conclusions: Depression treatment improves some, not all, subjective sleep disturbance components, with benefits diminishing after termination. Adjunctive interventions are likely needed to address the lingering components of sleep disturbance.

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Abbreviations: CVD, cardiovascular disease; CBT-D, cognitive-behavioral therapy for depression; PHQ-9, Patient Health Questionnaire-9; PSQI, Pittsburgh Sleep Quality Index; SOL, sleep onset latency; TST, total sleep time; SE, sleep efficiency; DSM-5, Diagnostic and Statistical Manual of Mental Disorders 5th Edition; MDD, major depressive disorder; CBT-I, cognitive-behavioral therapy for insomnia; CRC, Clinical Research Center; CTSL, Clinical and Translational Science Institute; BtB, Beating the Blues; PST-PC, Problem-Solving Treatment in Primary Care; DCS, depression care specialist; PCP, primary care provider; SNRI, serotonin-norepinephrine reuptake inhibitors; SSRI, selective serotonin reuptake inhibitor.

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1. Introduction

Depression is a highly prevalent, chronic, and persistent mental health condition that leads to loss of life, functional impairment, and economic burden across the world [1–5]. It is comprised of both cognitive/affective and somatic symptom clusters [6,7]. The cognitive/affective depressive symptoms are depressed mood, anhedonia, feelings of worthlessness or excessive guilt, difficulty concentrating or indecisiveness, psychomotor disturbances, and recurrent thoughts of death or suicidal ideation, whereas the somatic depressive symptoms are appetite/weight changes, fatigue, and sleep disturbance.

Among these symptoms, sleep disturbance is one of the most frequently endorsed. An estimated 71 % of people with major depressive disorder (MDD) report it as a main presenting symptom [8], perhaps because depression predicts future sleep disturbance [9], and sleep disturbance (specifically, insomnia) predicts future depression [10]. In people with depression, there is heterogeneity in the presentation of sleep disturbance. Frequently reported symptoms include difficulty falling asleep, frequent night awakenings, nightmares, early morning awakenings, nonrestorative sleep, and decreased total sleep time [11, 12]. Sleep disturbance may also drive some of the physical health ramifications of depression [13], as it has been associated with an increased risk of diabetes, cardiovascular disease (CVD), and other chronic conditions [14–19].

Like many health conditions, sleep disturbance disproportionately impacts marginalized groups, including Black people and those with lower socioeconomic status (SES) [20]. One study utilizing both objective and subjective sleep measures found that Black adults had shorter sleep duration, longer sleep onset latency, and poorer sleep efficiency than White and Asian adults combined, but no difference in subjective sleep quality was observed [21]. These findings are further supported by a meta-analysis of 14 studies that found that Black adults have shorter sleep duration and poorer sleep continuity than White adults [22]. Similarly, a meta-analysis of 19 studies with objective sleep measures revealed that lower SES is associated with shorter sleep duration, longer sleep onset latency, greater sleep fragmentation, and poorer sleep efficiency [23].

While it is well documented that cognitive-behavioral therapy for insomnia (CBT-I) improves depressive symptoms [24,25], few studies have examined the effect of evidence-based treatments for depression on sleep disturbance [26–28]. One influential study found that, in MDD patients with baseline sleep disturbance recruited from a mental health care center, over half continued to have sleep disturbance after achieving depression remission following treatment with either cognitive-behavioral therapy for depression (CBT-D) or antidepressants [28]. Thus, depression treatment may improve sleep disturbance in some individuals; however, further research is needed to confirm these results, extend these findings to other clinical settings, and characterize the magnitude of intervention effects. In addition, little is known about the effect of depression interventions on specific sleep components. The Pittsburgh Sleep Quality Index (PSQI) [29], a widely utilized scale assessing sleep disturbance, is well suited for examining specific subjective sleep components, including sleep onset latency (SOL), total sleep time (TST), sleep efficiency, sleep quality, sleep medication use, sleep disturbances, and daytime dysfunction.

To help fill these knowledge gaps, we conducted a secondary analysis of the eIMPACT randomized controlled trial, which compared our modernized collaborative care intervention for depression (internet cognitive-behavioral therapy [CBT], telephonic CBT, and/or select antidepressants) to usual primary care for depression (primary care providers supported by embedded behavioral health clinicians and psychiatrists) in 216 primary care patients with depression from a safety net healthcare system. The objectives of the present study are: (1) to determine the effect of our depression intervention on individual components of subjective sleep disturbance, (2) to examine race, education, and income as moderators of intervention effects on components of

subjective sleep disturbance, and (3) to assess associations between changes in depressive symptoms and changes in components of subjective sleep disturbance.

2. Materials and methods

2.1. Study design

This study is a secondary analysis of the eIMPACT trial conducted from 2015 to 2020 [30]. This trial was a 24-month, phase II, single-center, assessor blinded, parallel-group randomized controlled trial. The primary aim of the eIMPACT trial was to examine the effect of successful depression treatment on CVD risk biomarkers. Of relevance to the present study, participants completed the same battery of self-report scales at the pre-treatment visit, mid-treatment (6-month) call, post-treatment (12-month) visit, and follow-up (24-month) call. After the pre-treatment assessments, participants were randomized 1:1 stratified by age group (50–59 years, ≥60 years) and sex (male, female) to 12 months of modernized collaborative care for depression (eIMPACT intervention) or usual primary care for depression (comparator). Full details regarding trial methods are reported elsewhere [30]. The trial was approved by the Indiana University Institutional Review Board and the Eskenazi Health Research Committee. As previously reported in the main outcomes paper for this trial [30], the eIMPACT intervention, versus usual primary care for depression, yielded statistically significant ($p < 0.01$), moderate-to-large (Hedges' $g = -0.65$), and clinically meaningful (43 % responders vs. 17 % responders) improvements in depressive symptoms at 12 months.

2.2. Participants

Two-hundred sixteen participants were recruited from the primary care clinics of Eskenazi Health, a safety net healthcare system in Indianapolis, IN. Inclusion criteria required patients to be ≥ 50 years, have current depression (defined as Patient Health Questionnaire-9 [PHQ-9] score ≥10 [31] and a PHQ-9 depressive disorder diagnosis [which requires depressed mood and/or anhedonia] [32], and have elevated CVD risk (defined as ≥1 [if ≥60 years] or ≥2 [if 50–59 years] of the following CVD risk factors: hypertension, hypercholesterolemia, diabetes, or current smoking). Exclusion criteria were clinical CVD, HIV/AIDS, chronic kidney disease, active cancer or current cancer treatment, current pregnancy, continuous treatment for a systemic inflammatory condition in the past 3 months, current use of anticoagulant medications, severe cognitive impairment, bipolar or psychotic disorder, acute risk of suicide, and ongoing depression treatment with a psychiatrist outside of Eskenazi Health. Patients unable to understand English were also not eligible, because the intervention was only available in English.

2.3. Treatment groups

The eIMPACT intervention is a modernized version of the collaborative care approach of the IMPACT trial [33]. It consists of a multidisciplinary team delivering evidence-based depression treatments in line with patient preference using a stepped, flexible, treat-to-target approach. The IMPACT intervention was modernized by adding an internet CBT-D program called Beating the Blues US (BtB; Workpartners UPMC) and telephonic Problem-Solving Treatment in Primary Care (PST-PC). Additionally, the other psychological components (psychoeducation, behavioral activation, and antidepressant adherence support) were delivered by phone or FaceTime, and the antidepressant algorithm was optimized for CVD risk reduction. The remaining components of the IMPACT intervention were unaltered. The intervention team consisted of a Master's-level depression clinical specialist (DCS), a supervising psychiatrist, a primary care liaison, and the participants' usual primary care providers (PCPs).

BtB is an efficacious internet CBT-D for depression and/or anxiety

[34–36]. It utilizes an interactive, multimedia platform to deliver eight weekly sessions, with similar content and structure to face-to-face CBT-D. Participants were instructed to complete one BtB session per week sessions from a self-chosen location with internet access (e.g., home or work) and received weekly support from the DCS via phone.

PST-PC is an effective CBT-D developed for primary care [37,38]. The manualized intervention consists of eight weekly sessions, during which patients learn the seven problem-solving steps and how to apply them to current problems contributing to their depression. The DCS was certified in PST-PC and delivered all sessions by phone, which has been shown to be efficacious [39].

The antidepressant algorithm followed the IMPACT intervention manual guidelines [40], with our psychiatrist making updates to dosing and titrating in line with current standards. The algorithm was optimized for CVD risk reduction by restricting the IMPACT medication list to selective serotonin reuptake inhibitors (SSRIs), duloxetine, bupropion, and mirtazapine, which are FDA-approved antidepressants that are the safest from a cardiovascular perspective [41]. The intervention team made antidepressant recommendations, which the DCS communicated to participants and PCPs. PCPs wrote all prescriptions, and the intervention team and PCPs collaboratively managed pharmacotherapy.

The intervention process followed the IMPACT manual with some modifications [40]. To end the pre-treatment visit, participants met with the DCS over FaceTime to review depression education materials, begin behavioral activation [37], and schedule the initial telephonic visit. During the initial telephonic visit, the DCS completed an assessment interview and discussed treatment preferences and options. New participant cases were presented to the intervention team every two weeks by the DCS, and the team formulated a Step 1 treatment plan. Step 1 treatment was 2–3 months of CBT-D or an antidepressant consistent with patient preference. BtB was the first-line CBT-D, and telephonic PST-PC was the second-line CBT-D. SSRIs were the first-line antidepressants, and other medications were second-line antidepressants. The DCS monitored treatment progress for 12 months and staffed each case with the intervention team at least every 3 months. During active treatment, DCS contacts (typically 30 min by phone) occurred every 1–2 weeks. These contacts included assessing depressive symptoms, continuing behavioral activation, delivering CBT-D (if prescribed), and supporting antidepressant adherence (if taking an antidepressant). Participants who achieved remission [33] developed a relapse prevention plan with the DCS and would receive follow-up calls from the DCS every 2–4 weeks. If remission was not achieved after Step 1, a Step 2 treatment was delivered for an additional 2–3 months. Step 2 treatment involved augmenting Step 1 treatment with CBT-D or an antidepressant or switching to another CBT-D or antidepressant. If remission was not achieved after Step 2, a Step 3 treatment was delivered consisting of additional CBT-D and/or adjustments to the antidepressant regimen and, if indicated, a phone evaluation with the trial psychiatrist.

The usual care group, modeled after the comparator of the IMPACT trial [33], consisted of 12 months of usual primary care for depression. The Eskenazi Health primary care clinics use a team care approach for behavioral health issues, with PCPs supported by embedded Master's-level behavioral health clinicians and affiliated psychiatrists available for brief counseling and antidepressant medication management. To end the pre-treatment visit, participants were informed of their depression diagnosis, were provided with a list of local mental health services and were encouraged to follow-up with their usual PCP regarding their depression. Participants' PCPs were notified of their patient's depressive disorder and group assignment via a letter or EHR message, and they were encouraged to work with the participant to address their depression with no restrictions on the care they could provide.

2.4. Measures

2.4.1. Subjective sleep components

Subjective sleep components over the past month were assessed

using the PSQI [29]. This scale consists of 19 items that can be used to compute a PSQI global score and seven component scores. Given that many of the PSQI component scores collapse continuous data into a variable ranging from 0 to 3, we did not use them, except for sleep quality and sleep medication use. Instead, we used the continuous sleep component data captured by the PSQI as described below to retain the most information possible. The PSQI has demonstrated good test-retest reliability and criterion validity [29,42].

2.4.1.1. Overall sleep quality. To assess overall sleep quality, we utilized the PSQI global score, which is calculated by summing the seven PSQI component scores. Scores range from 0 to 21, with higher scores indicating poorer sleep quality.

2.4.1.2. Sleep onset latency (SOL). To quantify SOL, we used responses to PSQI item 2, which asks: "During the past month, how long (in minutes) has it usually taken you to fall asleep each night?" This is also the continuous data used to calculate the PSQI component 2 (sleep latency) score.

2.4.1.3. Total sleep time (TST). To quantify TST, we used responses to PSQI item 4, which asks: "During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spend in bed.)" This is also the continuous data used to compute the PSQI component 3 (sleep duration) score. We then examined the number of participants falling into the following categories based on the American Academy of Sleep Medicine's recommendations [43]: shorter than optimal (<7 h; $n = 172$), optimal (7–9 h; $n = 29$), and longer than optimal (>9 h; $n = 8$) at pre-treatment. Due to the low count for the longer than optimal category, we excluded these eight participants from analyses examining total sleep time so that improvements were only in one direction (i.e., increases).

2.4.1.4. Sleep efficiency. To calculate sleep efficacy, we first computed each participant's time in bed by subtracting the time to bed captured by PSQI item 1 ("During the past month, when have you usually gone to bed at night?") from the time out of bed captured by PSQI item 3 ("During the past month, when have you usually gotten up in the morning?"). We divided TST from PSQI item 4 by the calculated time in bed and set a maximum possible value of 100 %. This is also the continuous data used to calculate the PSQI component 4 (habitual sleep efficiency) score.

2.4.1.5. Subjective sleep quality. To measure sleep quality, we computed the PSQI component 1 (subjective sleep quality) score. Scores range from 0 to 3, with higher scores reflecting poorer sleep quality.

2.4.1.6. Sleep medication use. To assess sleep medication use, we calculated the PSQI component 6 (use of sleep medication) score. Scores range from 0 to 3, with higher scores indicating more frequent sleep medication use.

2.4.1.7. Sleep disturbances. To quantify sleep disturbances, we summed responses to PSQI items 5b–5j. Scores range from 0 to 27, with higher scores indicating a greater frequency of sleep disturbances. This is also the continuous data used to compute the PSQI component 5 (sleep disturbances) score.

2.4.1.8. Daytime dysfunction. We calculated a daytime dysfunction score by adding responses to item 8 ("During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?") and item 9 ("During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?"). Scores range from 0 to 6, with higher scores indicating greater daytime dysfunction. This is also the continuous data used to compute the PSQI component 7 (daytime dysfunction) score.

2.4.2. Candidate moderators

Race, education, and income were assessed by standard questions at the pre-treatment visit. Race was measured by asking participants, “Which race do you most identify with or consider yourself to be?” The original seven response categories (see Table 1) were recoded into a dichotomous variable (0 = White, 1 = Black, other racial groups set to missing) due to low frequencies (all $n_s \leq 8$) in the categories that were

Table 1
Baseline Characteristics of Participants by Treatment Group.

	All (N = 216)	Intervention (n = 107)	Usual Care (n = 109)	p value
Sociodemographic Factors				
Age, years, <i>M</i> (<i>SD</i>)	58.7 (5.7)	58.5 (6.0)	58.9 (5.4)	0.62
Sex, <i>n</i> (%)	169 (78.2)	83 (77.6)	86 (78.9)	0.81
Female	47 (21.8)	24 (22.4)	23 (21.1)	
Male				
Race, <i>n</i> (%)	1 (0.5)	0 (0.0)	1 (0.9)	0.70 ^a
American Indian/ Alaskan Native	1 (0.5)	1 (0.9)	0 (0.0)	
Asian	107 (49.5)	56 (52.3)	51 (46.8)	
Black/African American	0 (0.0)	0 (0.0)	0 (0.0)	
Native Hawaiian/Other Pacific Islander	97 (44.9)	45 (42.1)	52 (47.7)	
White	8 (3.7)	4 (3.7)	4 (3.7)	
Multi-Racial Other Race	2 (0.9)	1 (0.9)	1 (0.9)	
Ethnicity, <i>n</i> (%)	10 (4.6)	5 (4.7)	5 (4.6)	0.98
Hispanic or Latino/a/x Not Hispanic or Latino/ a/x	206 (95.4)	102 (95.3)	104 (95.4)	
Education, years, <i>M</i> (<i>SD</i>)	12.8 (2.3)	13.1 (2.5)	12.6 (2.0)	0.09
Annual Household Income, <i>n</i> (%)	98 (45.6)	44 (41.1)	54 (50.0)	0.04
< \$10,000	37 (17.2)	16 (15.0)	21 (19.4)	
\$10,000–\$14,999	45 (20.9)	21 (19.6)	24 (22.2)	
\$15,000–\$24,999	25 (11.6)	18 (16.8)	7 (6.5)	
\$25,000–\$39,999	10 (4.7)	8 (7.5)	2 (1.9)	
\$40,000 +				
Depressive Symptoms				
PHQ–9 Total Score (possible range: 0–27), <i>M</i> (<i>SD</i>)	15.1 (5.0)	14.7 (5.2)	15.4 (4.8)	0.33
Subjective Sleep Components				
PSQI Global Score (possible range: 0–21), <i>M</i> (<i>SD</i>)	12.6 (3.9)	13.0 (3.8)	12.2 (4.0)	0.17
Sleep Onset Latency, minutes/night, <i>M</i> (<i>SD</i>)	74.8 (61.7)	79.1 (67.8)	70.4 (54.7)	0.31
Total Sleep Time, hours/ night, <i>M</i> (<i>SD</i>)	5.4 (1.8)	5.2 (1.7)	5.6 (1.8)	0.11
Sleep Efficiency, %, <i>M</i> (<i>SD</i>)	72.2 (21.8)	71.9 (22.4)	72.6 (21.2)	0.83
Subjective Sleep Quality Score (possible range: 0–3), <i>M</i> (<i>SD</i>)	2.0 (0.9)	2.0 (0.8)	2.0 (0.9)	0.81
Sleep Medication Use Score (possible range: 0–3), <i>M</i> (<i>SD</i>)	1.3 (1.4)	1.4 (1.4)	1.1 (1.3)	0.07
Sleep Disturbances Score (possible range: 0–27), <i>M</i> (<i>SD</i>)	14.9 (5.1)	14.5 (4.8)	15.3 (5.2)	0.23
Daytime Dysfunction Score (possible range: 0–6), <i>M</i> (<i>SD</i>)	2.6 (1.4)	2.6 (1.5)	2.6 (1.3)	0.99

Note. All variables have complete data except (*n*): income (215), PSQI global score (193), sleep onset latency (211), total sleep time (209), sleep efficiency (200), subjective sleep quality score (215), sleep medication use score (214), sleep disturbances score (205), and daytime dysfunction score (213). *P* values are from independent-samples *t* tests for continuous variables and χ^2 tests for categorical variables comparing the intervention and usual care groups. PHQ–9 = Patient Health questionnaire-9. PSQI = Pittsburgh Sleep Quality Index.

^aDue to some low cell counts, race was recoded into a 3-level variable (Black/African American, White, other race) prior to conducting the χ^2 test.

not White and Black/African American. We decided to set to missing the other racial groups, as the samples sizes were not sufficient to examine moderation. Education was measured by asking participants, “What was the last grade or level of school you completed?” A continuous years of education variable was calculated from the responses. Annual household income was collected by asking participants, “What is your total household income?” The original eight response categories were collapsed into a 5-level variable (0 = <\$10,000/year, 1 = \$10,000–\$14,999/year, 2 = \$15,000–\$24,999/year, 3 = \$25,000–\$39,999/year, 4 = >\$40,000/year) due to low frequencies in the high-income categories (all $n_s < 2$).

2.4.3. Depressive symptoms

Depressive symptoms were assessed using the reliable and valid Patient Health Questionnaire-9 (PHQ-9) [31,32]. Total scores range from 0 to 27, with higher scores indicating greater depressive symptoms.

2.5. Statistical analysis

All variables were normally distributed except for SOL [44]. A $\log_{10}$ transformation was applied to SOL to normalize its distribution. Independent-samples *t* tests (for continuous variables) and χ^2 tests (for categorical variables) were run to identify any treatment group imbalance (defined as $p < 0.10$) on participant characteristics at baseline. All subsequent models included baseline characteristics exhibiting group imbalance as covariates.

To achieve this study’s objectives, a series of mixed-effects models were performed using the *lm4* package [45] in R 4.1.2 [46]. Three sets of models were tested for each outcome. First, to identify the appropriate change trend (i.e., appropriate functional form for the time effect) for the outcome over the four time points (baseline, 6 months, 12 months, and 24 months), unconditional linear and quadratic models were fit with time centered at baseline (time was coded as 0, 1, 2, 4). The two models were compared via a likelihood ratio test. The quadratic model was selected if the test was significant, indicating a better fit of the quadratic model. Otherwise, the linear model was selected due to its parsimony. Of note, a linear change trajectory is determined by two parameters: intercept (baseline status) and linear slope (change per 6 months over time), and a quadratic change trajectory is captured by three parameters: intercept (baseline status), linear slope (instantaneous slope at baseline), and quadratic slope (change rate of the instantaneous slope over time) [47,48].

Second, to examine intervention effects on changes over time for the outcomes, we added treatment group (0 = usual care, 1 = intervention) to the selected model to predict individual intercepts and slopes. To reveal the pattern of the intervention effects, we plotted the estimated mean change trajectories for both groups and each outcome. Additionally, we reran the models with time centered at different time points to test and calculate the effect size for the treatment group difference at each time point. Specifically, Cohen’s *d*s were computed with baseline group differences in the outcome adjusted (d_{adj}), where $d_{adj} = (D_t - D_1)/SD_{Pool}$. D_t is the estimated group mean difference at a target time point, D_1 is the estimated group mean difference at baseline, and SD_{Pool} is the pooled standard deviation of the outcome at baseline [49].

Third, race, education, and income were entered into each model to examine whether they moderate the intervention effects. In all models, random intercepts and slopes were included when possible (i.e., without causing convergence problems). Full information maximum likelihood estimation method was used to estimate models, which accommodates missing data and allows for comparisons of nested models with different fixed effects and variance components. A two-tailed $p < 0.05$ was considered statistically significant.

Finally, to assess associations between changes in depressive symptoms and components of subjective sleep disturbance during the treatment period, we calculated Pearson correlations between pre- to post-treatment change scores for the PHQ-9 total score and each subjective

sleep component across the full sample. Arithmetic change scores were computed by subtracting pre-treatment values from post-treatment values. Correlation effect sizes were interpreted using Cohen's guidelines [50], where $r = 0.10$ is small, $r = 0.30$ is medium, and $r = 0.50$ is large.

3. Results

3.1. Participant characteristics

As shown in Table 1, our sample included all 216 participants enrolled in the eIMPACT trial – 107 randomized to the intervention group and 109 to the usual care group. The mean age was 59 years. The sample was almost evenly split between Black adults and White adults. The mean years of education was 12.9, which equates to slightly above

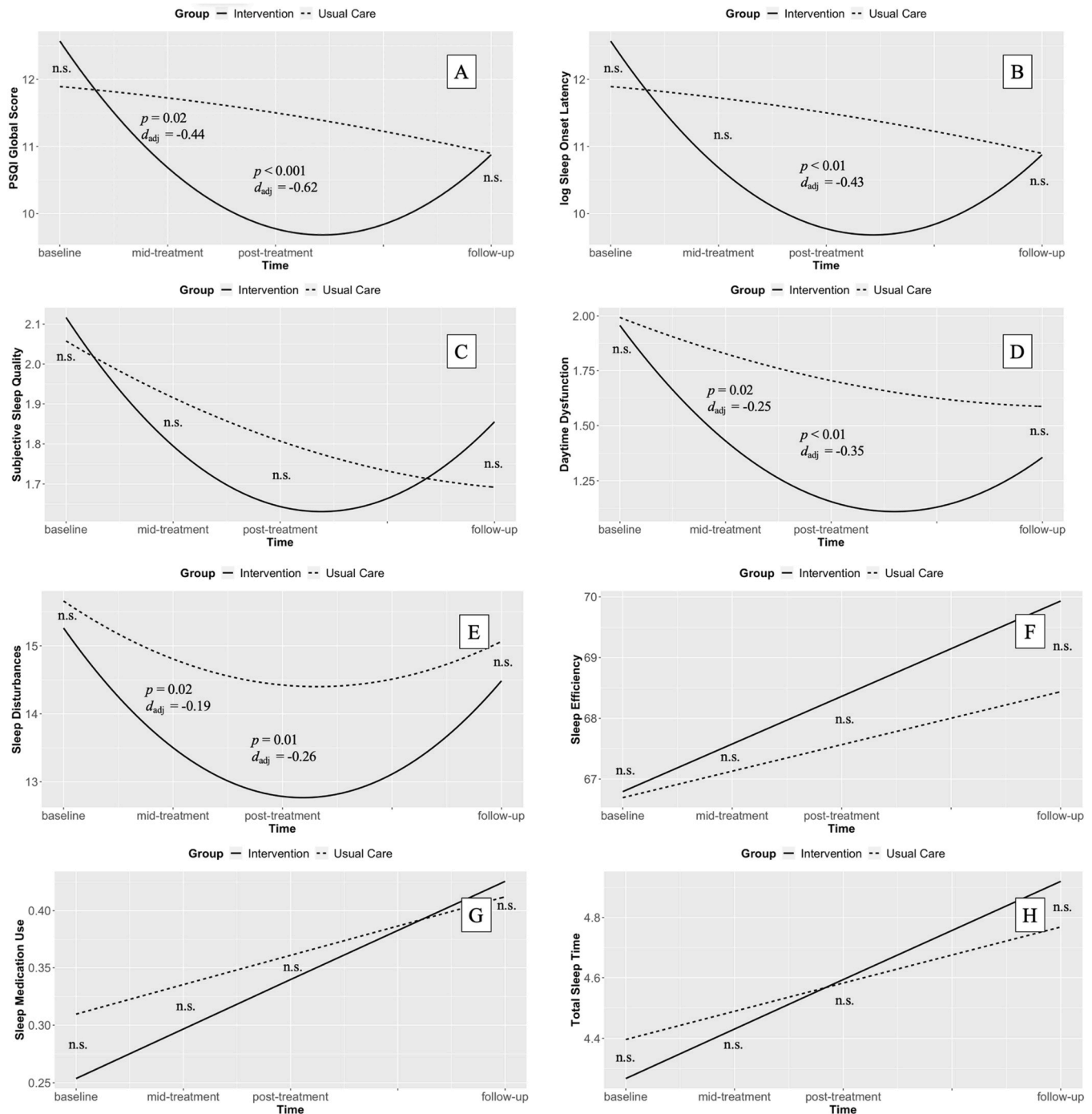


Fig. 1. Effect of the eIMPACT intervention, versus usual primary care for depression, on subjective sleep components across 24 months. Outcomes were assessed at pre-treatment (baseline), mid-treatment (6-months), post-treatment (12-months), and follow-up (24-months). The graphs were created based on quadratic mixed-effects models for all outcomes except for total sleep time, sleep efficiency, and sleep medication use score. For these three outcomes, linear mixed effects models were used. All models were adjusted for baseline imbalance in education, income, and sleep medication use. PSQI = Pittsburgh Sleep Quality Index; n.s. = not significant. $N = 216$.

high school graduate/GED. The sample also had a good representation of adults with lower income, as 46 % reported a household income of less than \$10,000/year. The mean PHQ-9 total score was 15.1, falling in the moderately severe depression range. The mean PSQI global score was 12.6, which is well above the recommended cut point of 5 distinguishing between good and poor sleepers [29]. Of the 193 participants with PSQI global score data, 187 (97 %) qualified as poor sleepers.

Baseline characteristics of participants were balanced across the treatment groups except for education, income, and PSQI sleep medication use score (see Table 1). The intervention group had somewhat higher education, income, and sleep medication use than the usual care group. Accordingly, all subsequent models included these three characteristics as covariates.

3.2. Effect of eIMPACT intervention on the change of subjective sleep components

Quadratic models fit the data better for all outcomes except for TST, SE, and sleep medication use score. As illustrated in Fig. 1, the eIMPACT intervention, versus usual primary care for depression, significantly influenced change across 24 months in four of the subjective sleep components: PSQI global score, SOL, subjective sleep quality score, and daytime dysfunction score. Significant treatment group effects on both linear and quadratic slopes were detected for each of these outcomes, indicating a significant change in these outcomes across the 24 months (see Table 2). For all these outcomes, the intervention group exhibited greater improvements than the usual care group. In contrast, the eIMPACT intervention did not significantly influence change in TST, sleep efficiency, sleep disturbances score, or sleep medication use score across the 24 months (see Table 2).

To better characterize the treatment group effect observed across the 24 months, post-hoc comparisons at each time point were conducted. As shown in Fig. 1, the intervention group exhibited significantly greater improvement at post-treatment for PSQI global score, SOL, sleep disturbances score, and daytime dysfunction score. For PSQI global score, sleep disturbances score and daytime dysfunction score, these significant intervention effects were also present at mid-treatment. No intervention effects were observed for any of the subjective sleep components at follow-up. Examination of the effect sizes (d_{adj}) reported in Fig. 1

Table 2
Treatment Group Effects on the Linear and Quadratic Slopes for Each Outcome Across 24 Months.

Outcome	Treatment Group Effect on Linear Slope		Treatment Group Effect on Quadratic Slope	
	B (SE)	p	B (SE)	p
PSQI Global Score	-2.23 (0.48)	< 0.001	0.51 (0.11)	< 0.001
Sleep Onset Latency	-0.15 (0.05)	< 0.01	0.03 (0.01)	< 0.01
Total Sleep Time	0.07 (0.06)	0.27	—	—
Sleep Efficiency	0.35 (0.87)	0.69	—	—
Subjective Sleep Quality Score	-0.25 (0.11)	0.03	0.07 (0.03)	0.01
Sleep Medication Use Score	0.02 (0.04)	0.69	—	—
Sleep Disturbances Score	-1.20 (0.61)	0.05	0.29 (0.15)	0.05
Daytime Dysfunction Score	-0.46 (0.19)	0.02	0.10 (0.05)	0.02

Note. All analyses were run on N = 216 except total sleep time which excluded participants with longer than optimal sleep time (n = 208). Values are from quadratic mixed-effects models for all outcomes except for total sleep time, sleep efficiency, and sleep medication use score. For these three outcomes, linear mixed-effects models were used. All models were adjusted for baseline imbalance in education, income, and sleep medication use. B and SE represent estimated unstandardized coefficients and standard errors, respectively. PSQI = Pittsburgh Sleep Quality Index.

reveals that the intervention group advantage increased over the intervention period, peaked at post-treatment, and decreased over the follow-up period for all the variables with significant intervention effects. At post-treatment, the group difference was moderate for PSQI global score ($d_{adj} = -0.62$) and SOL ($d_{adj} = -0.43$), small to moderate for daytime dysfunction score ($d_{adj} = -0.35$), and small for sleep disturbances score ($d_{adj} = -0.26$). No significant differences were found at examined time points for subjective sleep quality score.

3.3. Race, education, and income as candidate moderators of intervention effects

Moderation effects of race were not detected for the eIMPACT intervention effects on change in subjective sleep components across 24 months (see Supplemental Table 1). Similar results were found for the interactions of interest involving the education and income (see Supplemental Table 2 and 3, respectively). A single significant interaction emerged between education and treatment group on the linear slope for sleep efficiency. However, due to the absence of similar effects across other outcomes, and the high number of interaction tests conducted, this finding should be interpreted with caution.

3.4. Associations between changes in depressive symptoms and subjective sleep disturbance components

We observed several significant associations between pre- to post-treatment changes in depressive symptoms and subjective sleep disturbance components across the full sample (see Table 3). Notably, PHQ-9 changes had medium positive correlations with changes in PSQI global score, subjective sleep quality score, and daytime dysfunction score and small-to-medium positive correlations with changes in sleep onset latency and sleep disturbance, indicating that depressive improvements were associated with improvements in these sleep disturbance components. Similarly, PHQ-9 changes had a medium negative correlation with total sleep time, demonstrating that depressive improvements were associated with increases in sleep duration. Correlations between PHQ-9 changes and changes in sleep efficiency and sleep medication use score were small and not significantly significant.

4. Discussion

The objectives of the present study were to determine the effect of our depression intervention on subjective sleep disturbance components, to examine race, education, and income as moderators of intervention effects, and to assess associations between changes in depressive symptoms and changes in subjective sleep disturbance components. In this randomized controlled trial of racially and socioeconomically diverse primary care patients with depression, we found that the eIMPACT intervention yielded improvements in overall sleep quality, SOL,

Table 3
Pearson Correlations Between Pre- to Post-Treatment Changes in Depressive Symptoms and Subjective Sleep Disturbance Components.

Factors	r	p	95 % CI
PHQ-9 with PSQI Global Score	0.36	< 0.01	0.22, 0.49
PHQ-9 with Sleep Onset Latency	0.22	< 0.01	0.08, 0.35
PHQ-9 with Total Sleep Time	-0.27	< 0.01	-0.40, -0.13
PHQ-9 with Sleep Efficiency	-0.08	0.30	-0.22, 0.07
PHQ-9 with Subjective Sleep Quality Score	0.33	< 0.01	0.20, 0.44
PHQ-9 with Sleep Medication Use Score	0.07	0.32	-0.07, 0.21
PHQ-9 with Sleep Disturbances Score	0.18	0.02	0.03, 0.32
PHQ-9 with Daytime Dysfunction Score	0.31	< 0.01	0.18, 0.43

Note. All analyses were run on N = 216 except total sleep time, which excluded participants with longer than optimal sleep time (n = 208). Arithmetic change scores for all factors were computed post-treatment value minus pre-treatment value. CI = confidence interval. PHQ-9 = Patient Health Questionnaire-9. PSQI = Pittsburgh Sleep Quality Index.

subjective sleep quality, and daytime dysfunction across 24 months relative to usual primary care for depression. Across these outcomes, intervention effects were largest at post-treatment (with effect sizes ranging from small to moderate) and decreased considerably over the follow-up period. The observed improvements were not moderated by race, education, or income, suggesting that our depression intervention may be similarly effective at improving these sleep components for all examined groups. It should be noted, however, that the eIMPACT trial was not designed with moderation in mind and, thus, may be underpowered to detect such effects. Additionally, pre- to post-treatment improvements in depressive symptoms were associated with pre- to post-treatment improvements in overall sleep quality, subjective sleep quality, daytime dysfunction, sleep onset latency, sleep disturbance, and total sleep time, with effect sizes ranging from small to medium. In contrast to these results, we found no effects of the eIMPACT intervention on other subjective sleep components – i.e., total sleep time, sleep disturbances, sleep efficiency, or sleep medication use – across the 24 months. Collectively, our findings indicate that successful depression treatment improves some, but not all, subjective sleep components during active treatment; however, these effects diminish after termination of depression treatment.

The observed pattern of intervention effects on the subjective sleep components aligns well with the 3-factor model of the PSQI proposed by Cole et al. [51]. The three identified factors in that model are sleep efficiency (comprised of sleep duration and sleep efficiency), perceived sleep quality (comprised of sleep latency, subjective sleep quality, and sleep medication use), and daily disturbances (comprised of sleep disturbances and daytime dysfunction). Our results suggest that depression treatment: (a) is effective at improving most components of the perceived sleep quality and daily disturbances factors and (b) is not effective at addressing the sleep efficiency factor.

Our findings indicating a beneficial effect of depression treatment on some subjective sleep components are generally consistent with the small literature on this topic. Of note, our results extend prior findings to other clinical settings (i.e., primary care) and to marginalized groups disproportionately burdened by sleep disturbance. Blom et al. [26] found that internet CBT-D decreases insomnia symptoms measured by the Insomnia Severity Index (ISI), with a moderate-to-large effect size. However, because the individual insomnia components measured by the ISI were not examined, it is unclear which aspects drove this effect. In another study of people with depression, CBT-D led to improvements in overall sleep quality as indicated by the PSQI global score, with a moderate effect size [27]. Once again, individual sleep components were not examined. Likewise, a frequently cited paper by Carney et al. [28] reported that both CBT-D and pharmacotherapy for depression improved sleep disturbance in 49 % and 44 % of MDD patients with baseline sleep disturbance, respectively. Several subsequent studies have focused on the half of MDD patients in Carney et al.'s [28] study with residual sleep disturbance, which is important because residual sleep disturbance predicts future depressive episodes [52,53]. However, even CBT-D has depression response and remission rates of 42 % and 36 %, respectively [54], underscoring that it is common for a large percentage of patients to not respond to mental health treatments.

There are several plausible explanations for our finding that modernized collaborative care for depression improved some subjective sleep components. First, it is possible that our intervention's psychotherapies (internet CBT-D and PST-PC) led to reductions in daily stress and/or rumination and worry (e.g., via cognitive restructuring and problem-solving), which in turn improved specific sleep components (e.g., SOL) [55]. In addition, one of the sessions of the internet CBT-D we used has an optional sleep management module, which could also be responsible for some of observed improvements. Second, subjective sleep quality may have improved due to patients having more positive appraisals of their sleep following reductions in their depressive symptoms [56]. Third, the PSQI daytime dysfunction score reflects is comprised of items assessing enthusiasm during the day and the ability

to stay awake during activities such as driving, eating meals, or engaging in social activity. Because these items are closely tied to the cognitive/affective symptoms of depression (e.g., depressed mood and anhedonia), reductions in daytime dysfunction may more closely align with depression improvement in this context and less with sleep disturbance improvement.

In addition to the strengths of this study (randomized controlled trial design, diverse sample, use of advanced statistical methods, and the substantial intervention effect on depressive symptoms), there are also limitations worth discussing. First, the eIMPACT trial was not a priori powered to detect treatment group differences in subjective sleep disturbance components, and we did not apply a multiplicity correction due to the detrimental effects on statistical power. Consequently, the present results should be considered hypothesis generating and exploratory in nature, rather than confirmatory. Second, because objective sleep measures were not obtained in the eIMPACT trial, we could not quantify the impact of our intervention on these metrics. The PSQI is a strong assessment for subjective sleep complaints; however, the gold standard for assessing sleep disturbance is polysomnography or actigraphy, which were not obtained in this study. Future studies should acquire data for both subjective (via self-report) and objective (via actigraphy or polysomnography) measures of sleep components. Third, while our sample had a good proportion of Black adults and White adults, there was insufficient representation of other races to examine them in the moderation analyses. Similarly, due to the safety net healthcare setting of the eIMPACT trial, income in our sample was skewed toward the lower levels and had little representation of the higher levels. Fourth, given that the eIMPACT trial was not designed to test for moderation, our moderation analyses may be underpowered and, thus, could have failed to detect meaningful differences in intervention effects across racial groups, education levels, and income levels. Future studies should also examine such moderation effect to assess intervention efficacy in the sociodemographic groups most impacted by sleep disturbance.

In conclusion, we found that successful depression treatment improves some, but not all, subjective sleep components and that these beneficial effects diminish after treatment termination. Similarly, we found that improvements in depressive symptoms were associated with improvements in some, but not all, subjective sleep components. Our study (a) provides evidence for the utility of depression treatment for improving some subjective sleep components (i.e., SOL, subjective sleep quality, and daytime dysfunction) among diverse patients with depression and disturbed sleep and (b) clarifies which subjective sleep components (i.e., total sleep time, sleep disturbances, sleep efficiency, and sleep medication use) that may need to be more actively targeted with adjunctive intervention (i.e., CBT-I) depending on the specific patient presentation of disturbed sleep. Our findings also help explain the conflicting findings that depression treatment improves sleep disturbance and that sleep disturbance is a common residual symptom after depression treatment that contributes to depression recurrence. Future research should examine which specific sleep components are predictors of depression recurrence. It is our hope that this research will assist in providing symptom relief to patients experiencing both depression and sleep disturbance and give providers additional guidance regarding the strengths and limitations of available evidenced-based treatments.

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Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Jesse C. Stewart reports financial support was provided by National

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.xjmad.2025.100132](https://doi.org/10.1016/j.xjmad.2025.100132).

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