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## Network Intervention Analyses of cognitive therapy and behavior therapy for insomnia: Symptom specific effects and process measures

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### ABSTRACT

Cognitive therapy (CT) and behavior therapy (BT) are both effective for insomnia. In this study we applied Network Intervention Analysis to investigate specific effects of CT and BT on outcomes *and* process measures. The analysis was based on a randomized controlled trial comparing CT ( $n = 65$ ), BT ( $n = 63$ ) and cognitive behavioral therapy for insomnia ( $n = 60$ ; not included in this study). In the first networks, the separate items of the Insomnia Severity Index and sleep efficiency were included. In the second networks, the pre-specified process measures for BT and CT, sleep efficiency, and the sum-score of the Insomnia Severity Index were included. At the different time points, we found CT-effects on worry, impaired quality of life, dysfunctional beliefs, and monitoring sleep-related threats, and BT-effects on sleep efficiency, difficulty maintaining sleep, early morning awakening, time in bed, sleep incompatible behaviors and bed- and rise time variability. These observed effects of CT and BT were consistent with their respective theoretical underpinnings. This study provided new information on the mechanisms of change in CT and BT. In the future, this may guide us to the most effective treatment modules or even subsets of interventions.

Insomnia is a prevalent and debilitating disorder (Morin & Benca, 2012). People with insomnia have trouble falling and/or staying asleep. In order to fulfill DSM-5 diagnosis, the sleep disturbance also needs to be associated with impaired daytime functioning (American Psychiatric Association, 2013). People with insomnia often have impaired quality of life (Kyle et al., 2010) and are prone to developing other forms of psychopathology, such as depression (Baglioni et al., 2011).

The treatment of choice for insomnia is cognitive behavioral therapy (CBT-I; Qaseem et al., 2016; Riemann et al., 2017). CBT-I is an effective treatment (Edinger et al., 2021; van der Zweerde et al., 2019; van Straten et al., 2018) with a cognitive part and a behavioral part as its main components. In cognitive therapy (CT), the focus is on selective attention, misperception of sleep, and dysfunctional beliefs about sleep (Jansson-Frojmark & Norell-Clarke, 2018). Behavior therapy (BT) focuses on sleep restriction and/or stimulus control to fix rising times and regulate the circadian system (Bootzin et al., 1991; Spielman et al.,

1987). In sleep restriction, bedtime is restricted in order to increase sleep pressure (Maurer et al., 2018). In stimulus control, conditioning procedures are used in order re-associate the bed to sleeping.

Even though CBT-I is an effective treatment, still about 20–30% of patients do not show a clinical response to CBT-I, and ~60% do not remit (Morin & Benca, 2012), highlighting the need for improvement. To improve treatment, it is essential to have a better understanding of *how* the treatment works. Here, it is important to realize that CT and BT have different theoretical backgrounds. Cognitive theory proposes that it is necessary to break through the vicious cycles of excessive worry, selective attention, distorted perception, unhelpful beliefs, and safety behaviors (Harvey, 2002; Morin, 1993). BT is based on the idea that perpetuating factors need to be reduced, the bed needs to be re-associated with sleep, and the circadian cycle needs to be regulated (Bootzin et al., 1991; Spielman et al., 1987). Since CT and BT both have very different theoretical backgrounds, they may also have different

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mechanisms of change. More knowledge about these mechanisms of change may guide further treatment development. CBT-I is mostly investigated as a full package, but CT and BT are also effective in a stand-alone format (Harvey et al., 2007, 2014; Sunnhed et al., 2020). Indeed, studies have shown that CT and BT are equally effective (Harvey et al., 2014; Sunnhed et al., 2020), but BT may have a faster and CT a more sustained effect (Harvey et al., 2014).

In a secondary analysis of one of these two datasets (Sunnhed et al., 2020), we aimed to improve the understanding of the mechanisms of change of CT and BT (Blanken et al., 2021). We did so by employing Network Intervention Analysis (NIA; Blanken et al., 2019); a relatively new technique based on the network theory of mental disorders (Borsboom, 2017). NIA moves beyond merely analyzing the sum-scores of the outcomes: the method enables us to see what effects on specific symptoms emerge at separate time points. In these networks the interdependency among the symptoms is considered, such that direct and indirect effects can be distinguished. NIA thus allows us to estimate a network of symptoms at separate time points throughout treatment to see which symptoms are affected by either CT or BT.

In our earlier NIA-study (Blanken et al., 2021) we identified that halfway through treatment people in the BT condition showed symptom-specific effects on sleep efficiency and difficulty maintaining sleep. The CT condition showed symptom-specific effects on interference with daily functioning. More symptom specific effects were observed at post-test. Here, BT showed effects on sleep efficiency and dissatisfaction with sleep. CT showed effects on difficulty initiating sleep, early morning awakenings and worry about sleep (Blanken et al., 2021). These results were in line with the theoretical assumptions of both CT and BT. However, as these symptom-specific effects are based on a single sample, replicating these findings is essential to strengthen our understanding of the symptom-specific effects over the course of treatment of CT and BT.

For this current paper, we were offered the unique opportunity to replicate these analyses on what is, to our knowledge, the only other dataset available directly comparing the effects of BT and CT in insomnia. In this randomized controlled trial (RCT), CT and BT were compared with CBT (Harvey et al., 2014). Our first aim was to replicate our earlier study (Blanken et al., 2021). We focused on CT and BT exclusively in order to contrast CT and BT effects. We excluded the CBT condition, as this approach combines both treatments and it could have obscured the pure CT and BT effects. We followed the previous study and included all the ISI symptoms and sleep efficiency in the model. In line with our earlier findings, we expected that CT would directly affect the cognitive symptoms (e.g. worry), whereas BT would directly link to the behavioral symptoms (e.g. sleep efficiency).

In addition, the current dataset enabled us to go a step further than mere replication, as Harvey and colleagues also included process mediators in their study (Harvey et al., 2017). They predefined the following *cognitive* processes: worry, unhelpful beliefs, and monitoring for sleep-related threat; and the following *behavioral* processes: sleep-incompatible behaviors, bedtime variability (BTv), risetime variability (RTv), time in bed (TIB). They observed that behavioral processes mediated BT outcomes and cognitive processes mediated both CT and BT outcomes. In these analyses, however, the association among these processes themselves was not considered. As these different processes are supposed to be affected by a similar treatment, it is likely that they are themselves associated. If we do not take this interdependency into account, we cannot distinguish the unique effects each of these processes has on the experienced symptoms. With NIA we can investigate which specific mediators are affected by the treatments, while controlling for the interdependency among all processes. Therefore, the second aim of this study is to investigate on which mediators we observe differential effects between CT and BT. In line with earlier findings, we expected that CT would affect cognitive mediators and BT would affect behavioral mediators and possibly also the cognitive mediators.

## 1. Method

### 1.1. Design and sample

The data of the current project came from an RCT on the treatment of insomnia (Harvey et al., 2014). In this RCT 188 patients with insomnia were assigned to either CT ( $n = 65$ ), BT ( $n = 63$ ), or CBT ( $n = 60$ ). The participants in the trial were on average 47.7 years ( $SD = 12.6$ ) and had 15.5 years of education ( $SD = 3.3$ ); 117 (62.2%) were female and 13 (6.9%) were non-Caucasian. For the current paper, we excluded the CBT condition to get the clearest indication of the effects specific to CT and BT. Measurements were at pre-, mid-, and post-test treatment. Furthermore, there was a 6- and 12-month follow-up. Patients were recruited from two sites (University of California, United States and Université Laval, Canada) from March 2008 to November 2011. The original study was approved by the research ethics committees of the University of California (Berkeley) and the (Québec City) and registered at [clinicaltrials.gov](https://clinicaltrials.gov): NCT00869934.

Inclusion criteria were: (a) 25 years of age or older, and (b) meeting criteria for persistent insomnia. Exclusion criteria were: (a) progressive or unstable physical illness or neurological de-generative disease directly related to the onset and course of insomnia; (b) use of hypnotics and other medications known to alter sleep (patients on SSRI for at least 3 months were included); (c) evidence of sleep apnea, restless legs or periodic limb movements during sleep, or a circadian-based sleep disorder; (d) irregular sleep schedules; (e) current or past psychological treatment of insomnia within the past 5 years; (f) individuals consuming more than two alcoholic beverages or more than four caffeinated beverages per day were required to reduce their intake; and (g) a lifetime diagnosis of a psychotic or bipolar disorder or more than two lifetime episodes of major depressive disorder or an untreated current major depressive disorder or alcohol or drug abuse within the past year. See Harvey et al. (2014) for a more detailed description.

### 1.2. Treatments

Both CT and BT treatments consisted of eight weekly individual sessions of 45–60 min. Treatments were delivered by either licensed therapists or advanced graduate students in clinical psychology. Overlapping elements of the treatments were: a general overview of CBT, 3P model of insomnia, sleep diary, setting treatment goals, and sleep hygiene information.

In CT a broad range of cognitive maintaining factors were targeted. These factors included: sleep-related worry, selective attention, distorted perception, unhelpful beliefs and safety behaviors (Harvey et al., 2017). In addition, a minimum of four individually formulated behavioral experiments to test beliefs were included in the CT condition.

BT consisted of a combination of stimulus control procedures and sleep restriction. In sleep restriction, bedtime is restricted to the actual sleeping time whereafter it is gradually increased to the optimal sleeping time (Bootzin et al., 1991; Spielman et al., 1987).

### 1.3. Outcomes

For both the symptom-specific effects and the process measures, sleep efficiency and the ISI were used. The ISI was developed to assess insomnia severity. It consists of 7 items (range 0–4) where higher scores indicate more complaints (Bastien et al., 2001). The 7 items are: (a) difficulty initiating sleep (dis), (b) difficulty maintaining sleep (dms); (c) early morning awakenings (ema); (d) dissatisfaction with sleep (dissat); (e) interference with daily functioning (idf); (f) noticeability of impaired quality of life (niqol); (h) worry/distress about sleep (worry). In the first series of networks the separate items of the ISI were used. In the second series of analyses the sum-score of the ISI was used. Sleep efficiency was derived from the consensus sleep diary (Carney et al., 2012) where sleep efficiency is the percentage of time spent asleep of the total time in bed.

For the second aim additional process measures were included as mediators. CT mediators: 1) sleep-related worry was measured by the Anxiety and Preoccupation about Sleep Questionnaire (APSQ). The APSQ is a 10-item questionnaire where higher scores indicate more sleep-related worry (range 10–100; Tang & Harvey, 2004). 2) Dysfunctional beliefs were measured with the brief version of the Dysfunctional Beliefs and Attitudes about Sleep scale (DBAS-16), a 16-item version of the original DBAS and higher scores indicate more dysfunctional beliefs (range 0–10; Morin et al., 2007). 3) The tendency to focus on and monitor sleep-related threat was measured with the Sleep Associated Monitoring Index (SAMI). The SAMI is a 30-item self-report measure with higher scores indicating more monitoring (range 33–165; Semler & Harvey, 2004).

BT mediators were: 1) time in bed derived from the sleep diary; 2) bedtime- and rise variability from the sleep diary (individual standard deviation over 7-day period); 3) sleep incompatible behaviors, measured with the Sleep Behavior Rating Scale (SBRS). The SBRS consists of 20 items concerning sleep incompatible behaviors during the day, in the evening, in bed or around bed (range 20–100; Kazarian et al., 1979).

#### 1.4. Statistical analysis

First, to investigate the symptom-specific effects for CT and BT we used NIA (Blanken et al., 2019) and estimated, for each of the assessments, a Mixed Graphical Model (mgm; Haslbeck & Waldorp, 2020) including all items of the ISI and sleep efficiency (continuous) in addition to a binary treatment variable. As the networks are estimated on full datasets, we included all available data for each assessment, resulting in slight variations in sample size: 125, 113, 99, 97, 94, for the different assessments respectively. Given the relatively low sample size, we applied LASSO regularization to prevent the inclusion of spurious edges due to sampling variation (Epskamp & Fried, 2018) and in line with recent recommendations (Isvoranu & Epskamp, ). In addition, we checked the accuracy and stability of the estimated networks (Epskamp et al., 2018). See the supplementary materials for more details on the LASSO regularization and stability checks.

Second, to investigate the effects of CT and BT on the mediators, we repeated the procedures above, but now included the sum scores of the questionnaires reflecting the CT and BT mediators (i.e., APSQ, DBAS, SAMI, SBRS, time in bed, bedtime variability, risetime variability) as continuous variables, as well as the binary treatment variable. Again, the networks are estimated on complete datasets, resulting in the same slight variations in sample sizes (see above). All analyses were performed in R (version: 3.6.2) with the package *mgm* (version: 1.2–12).

#### 1.5. Interpretation

The estimated networks will be visualized and can be interpreted as follows. All variables are included as *nodes*, and all continuous variables will be presented as circles, whereas the binary treatment allocation variable will be included as a square. The variables will be connected by *edges* that represent de conditional dependence relations among them, i. e., the unique association between two variables after conditioning on all other variables in the network. Blue edges indicate positive associations, and red edges indicate negative associations. Since we are contrasting two active treatment conditions (CT vs BT), any edge between the treatment allocation variable and another symptom or mediator, will delineate a treatment-specific effect. Any effect in favor of CT will be represented by a green edge, and any effect in favor of BT will be represented by a yellow edge. It is important to keep in mind that any direct effects will specifically reflect *differences* between the two active treatments. Hence, if the two treatments affect a symptom or mediator similarly, this effect will not be represented in the network. Consequently, the absence of an edge does not indicate the absence of a treatment effect, but merely the absence of a *differential* treatment effect between CT and BT.

## 2. Results

### 2.1. First aim: differential effects on insomnia symptoms and sleep efficiency

In the Network Intervention Analysis on sleep efficiency and the items of the Insomnia Severity Index, we observed that at baseline, as expected, there were no differences between the CT and BT condition. At mid-treatment, CT showed a symptom specific effect on ‘worry’ (green line) while BT showed a symptom specific effect on ‘sleep efficiency’ and ‘difficulty maintaining sleep’ (yellow lines). At post-test only the effect of BT on ‘sleep efficiency’ remained, and at 6-month follow-up no symptom specific effects were observed. At 12-month follow-up, BT showed a differential effect on ‘sleep efficiency’ and on ‘early morning awakening’. CT linked to ‘impaired quality of life’. Please see Fig. 1 for the networks.

### 2.2. Second aim: differential effects on CT and BT processes

In the second series of Network Intervention Analyses we investigated the effect of treatment on CT and BT processes, including ISI sum score and sleep efficiency. As expected, there were again no baseline differences prior to treatment. At mid-treatment, in line with the a priori defined processes, CT showed a larger effect on ‘worry’ and ‘dysfunctional beliefs’ (green lines) and BT showed a larger effect on ‘time in bed’, ‘sleep incompatible behaviors’, ‘sleep efficiency’ and ‘risetime variability’ (yellow lines). At post-test, CT showed differential effects on ‘dysfunctional beliefs’ and ‘monitoring for sleep related threat’. BT had the same differential effects as mid-treatment, except it was now connected to ‘ISI’ instead of ‘sleep efficiency’. At 6-month follow-up, CT showed a differential effect for ‘monitoring for sleep related threat’ and BT for ‘sleep incompatible behaviors’ and ‘sleep efficiency’. At 12-month follow-up CT showed differential effects for ‘dysfunctional beliefs’; and BT for ‘sleep efficiency’ and ‘bedtime variability’. Please see Fig. 2 for the networks.

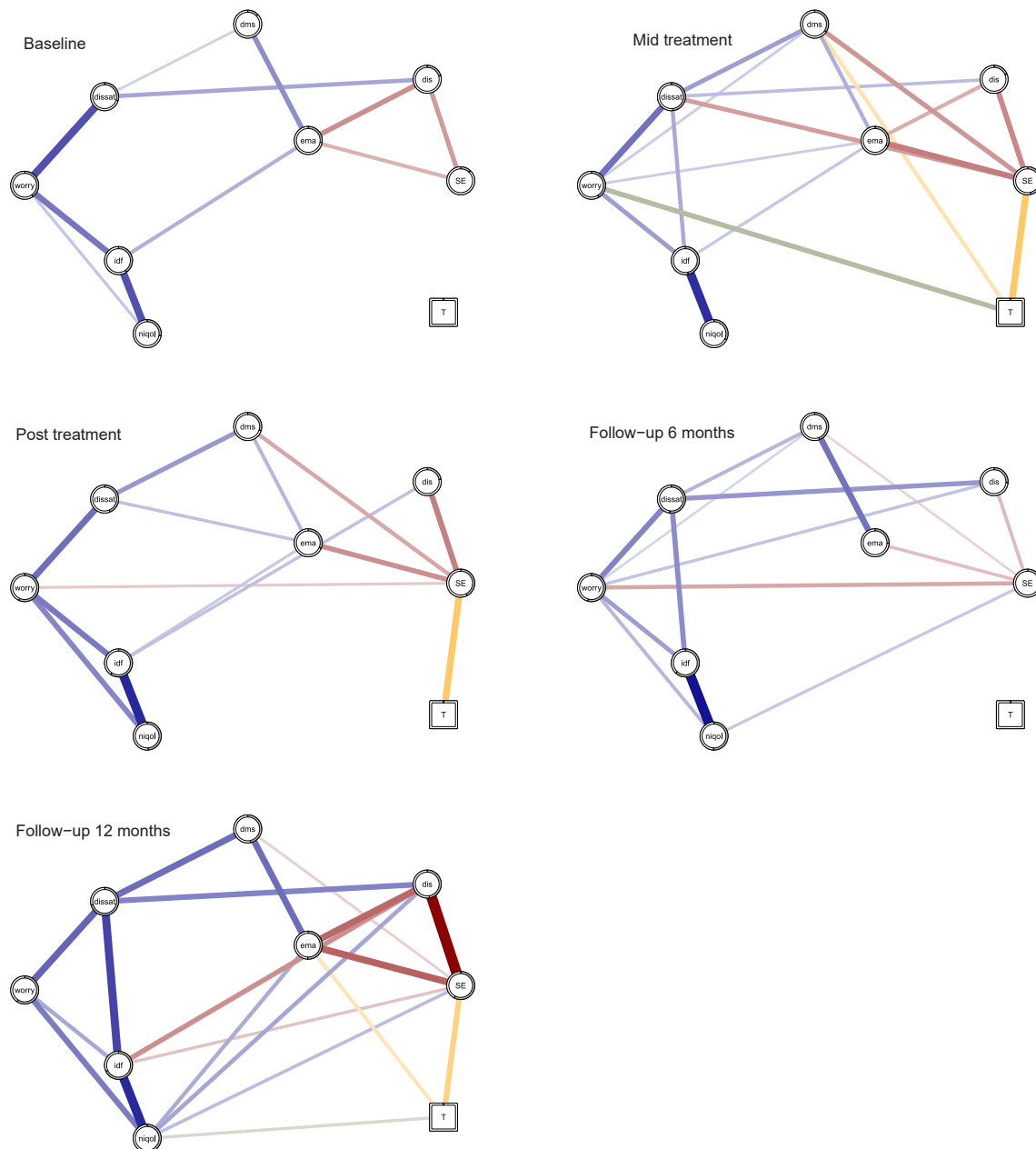
## 3. Discussion

In the current study we had two aims: 1) to replicate the earlier Network Intervention Analyses on the symptom-specific effects of CT and BT over time; and 2) to investigate the differential effects of CT and BT on pre-defined process measures.

For the first aim, the mid-treatment and post-treatment symptom-specific effects of CT and BT were in line with the theoretical assumptions of the treatments. Most markedly, CT showed a symptom specific effect on ‘worry’ and BT showed an effect on ‘sleep efficiency’. These BT results mirror the findings that were observed in week 4 of our previous study (Blanken et al., 2021). In the earlier study we did not observe CT effects mid-treatment, but at week 8 a CT effect on ‘interference with daily functioning’ did emerge. In both cases the findings are in line with what would be expected of the theoretical underpinnings (Bootzin et al., 1991; Harvey, 2002; Spielman et al., 1987).

For the second aim, we observed that on the process measures, BT only showed differential effects on behavioral processes and CT showed only differential effects on cognitive processes, exactly consistent with the theoretical models (Bootzin et al., 1991; Harvey, 2002; Morin, 1993; Spielman et al., 1987). Interestingly, contrary to the earlier findings (Harvey et al., 2017), BT had only connections with behavioral processes and no longer with cognitive process; possibly because the relationships among processes was now considered.

There are several limitations. First, this was a retrospective study. Knowing the efficacy outcomes may have induced bias in the interpretation of the results. Second, even though we know the starting point of the network (treatment node), analyses show partial correlations, not causality. Third, because the data were already collected, we were tied to available time-points. This meant that we only had a mid-treatment



*Note.* Please see the 'interpretation' section for an explanation of the observed relationships. Abbreviations: dis = difficulty initiating sleep; dms = difficulty maintaining sleep; ema = early morning awakenings; dissat = dissatisfaction with sleep; idf = interference with daily functioning; niqol = noticeability of impaired quality of life; worry = worry about sleep; SE = sleep efficiency; T = treatment. For each node, the proportion of explained variance by the other nodes in the network, i.e., the predictability, is visualized by a ring around each node: a completely filled ring indicates that 100% of the variance is explained, a completely empty ring corresponds to an explained variance of 0%.

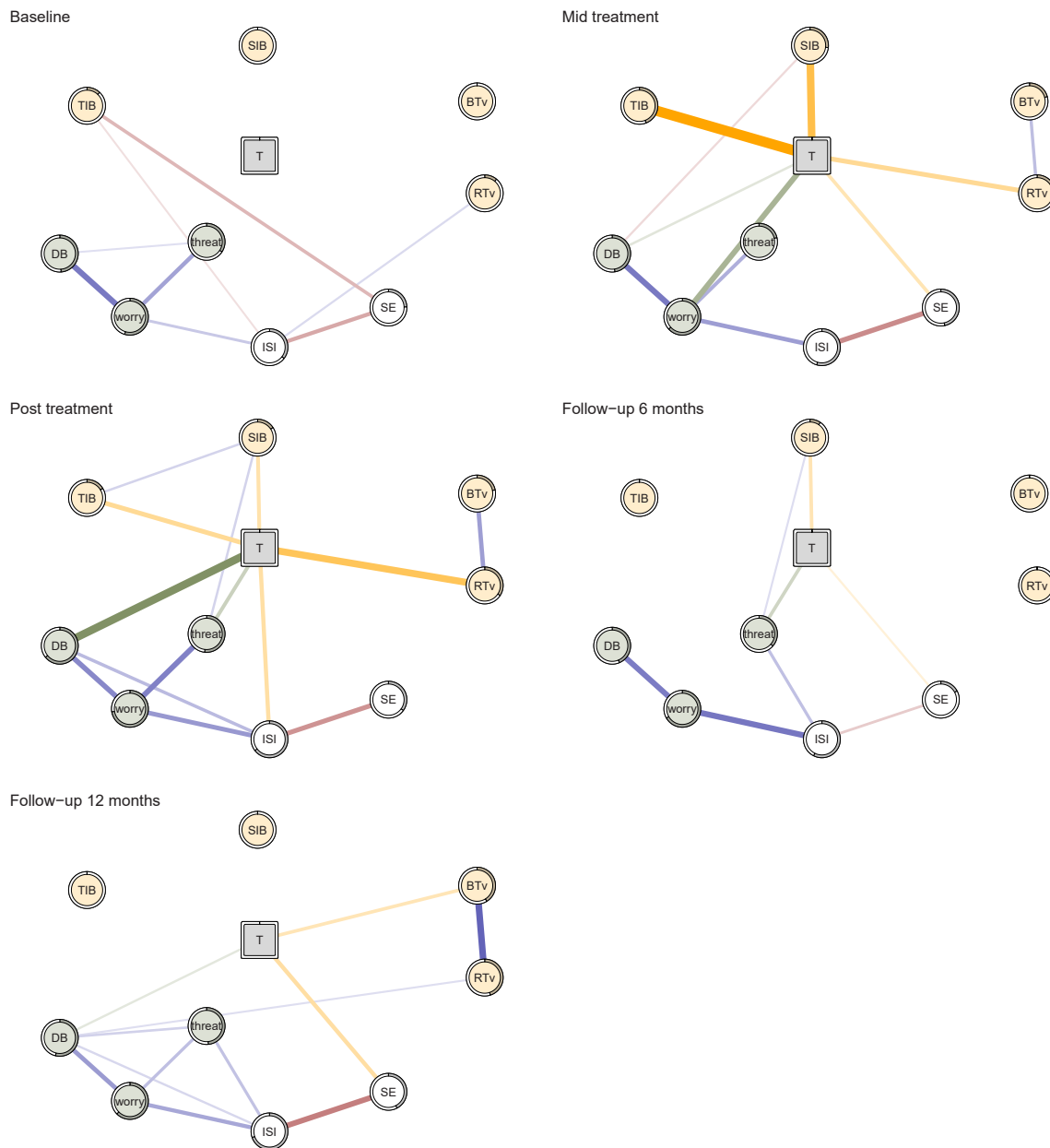
**Fig. 1.** Symptom specific effects of cognitive therapy (CT) and behavior therapy (BT) on insomnia symptoms and sleep efficiency.

measure and not bi-weekly measures as in the previous study. Fourth, the observations on the CT versus BT comparisons come only from two trials, potentially limiting generalizability.

Another important issue is that in both analyses we see associations in line with the theoretical models, but not *all* expected associations are observed. For instance, in the analyses on the process measures one would expect a mid-treatment BT effect for 'bedtime variability' (Fig. 2). Here it is important to realize that the observed effects were controlled for all the other effects in the networks. This means that both treatments

could still influence these variables but they may not differentiate between the treatments or may not survive controlling for the other effects. In this vein, these results may even be more interesting. In BT it appears risetime variability is of higher importance than bedtime variability. Here the variety of the bedtimes may be dependent on the variety of the risetime. A similar logic may apply to the CT process mediators, where the effect on the monitoring of sleep related threat (post-test) may partly follow from targeting worry and dysfunctional beliefs (mid-treatment) through their direct associations (i.e., positive links between





*Note.* Please see the 'interpretation' section for an explanation of the observed relationships. Abbreviations: BTv = Bedtime variability; DB = dysfunctional beliefs; ISI = Insomnia Severity Index; RTv = Risetime variability; SIB = Sleep incompatible behaviors; SE = sleep efficiency; threat = monitoring for sleep-related threat; TIB = Time in bed; T = treatment. For each node, the proportion of explained variance by the other nodes in the network, i.e., the predictability, is visualized by a ring around each node: a completely filled ring indicates that 100% of the variance is explained, a completely empty ring corresponds to an explained variance of 0%.

**Fig. 2.** Differential effects of cognitive therapy (CT) and behavior therapy (BT) on process measures.

dysfunctional beliefs, worry, and threat).

Even though not all the expected associations were observed it is striking that all associations found were in line with the theoretical assumptions. Apparently when other variables are controlled for, BT and CT are very much distinguishable along their theoretical lines. Worry came up as a distinguishable factor for CT in both the analyses on the ISI symptoms and on the process measures. This may mean that worry is affected first and from here the CT effect spreads throughout the network. It could also be that worry is a construct that is more easily and

rapidly targeted, whereas changes to dysfunctional beliefs may take some more time. These results tentatively support targeting worry first in CT because it may be an important or accessible hub in the CT network.

BT had very different discriminating factors than CT. In the ISI analysis, BT predominantly showed symptom-specific effects on sleep efficiency and difficulty maintaining sleep. These effects on sleep efficiency may serve as a manipulation check since sleep efficiency is directly targeted by the intervention. This may also apply to the time in

bed association with BT in the process measures. Interestingly, sleep efficiency at mid-treatment was, against our expectation, not related to time in bed. Another interesting observation is that the BT mediators are at no point connected to the ISI; BT may show its differential effect on the ISI through sleep efficiency.

A valuable next step would be to design a study to identify treatment-specific points of engagement. For instance, in CT, the effects of targeting either worry (e.g., by scheduled worry time) or dysfunctional beliefs first (by challenging misconceptions) could be tested. It would also be very informative to know what the specific effect of behavioral experiments are. Behavioral experiments concern planned experiential activities to rest the validity of beliefs and it would be interesting to know if these experiments influence specific process measures. For BT, we may build on studies such as a recent mechanistic trial that demonstrated that restricting time in bed was superior to only fixed bedtimes (Maurer et al., 2020). In this trial sleep timing was manipulated and at the same time vigilance was measured (Maurer et al., 2021). Trials like this could help in understanding how TIB, bed- and rising times are related to such outcomes. Another factor of interest is timing. In future studies, we could manipulate the moment at which modules are offered and form a science-based modular CBT-I system. Here, network analysis would aid us to detect symptom patterns in order to help with module selection.

In the current paper we have looked at CT and BT in isolation. In the future, this may guide us to the most effective treatment modules or even subsets of interventions (e.g., targeting worry vs beliefs) for specific insomnia patients. For now, we observed that the findings in both the model on the ISI symptoms and the model on the process measures are in line with the theoretical background of CT and BT. These findings helped us to establish a firmer empirical basis for the theoretical background of both BT and CT. This is important for the field of insomnia but also for the field of CBT in general.

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## Author's contribution

Jaap Lancee and Tessa Blanken developed the study concept. All authors contributed to the study design. Testing and data collection were performed by Allison Harvey, Charles Morin and Hans Ivers. Tessa Blanken performed the data analysis. Jaap Lancee drafted the paper, and Tanja van der Zweerde, Tessa Blanken and Allison Harvey provided critical revisions. All authors approved the final version of the paper for submission.

## Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Dr Morin reported the following: research support from Idorsia, Eisai, Lallemand Health, and Canopy Health; consultant/advisory board for Merck, Eisai, Pear Therapeutics, Sunovion, and Weight Watchers; royalties from Mapi Research Trust. The other authors reported no conflict of interest.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.brat.2022.104100>.

## References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Association.
- Baglioni, C., Battagliese, G., Feige, B., Spiegelhalter, K., Nissen, C., Voderholzer, U., ... Riemann, D. (2011). Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies. *Journal of Affective Disorders*, 135(1–3), 10–19. <https://doi.org/10.1016/j.jad.2011.01.011>
- Bastien, C. H., Vallières, A., & Morin, C. M. (2001). Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Medicine*, 2, 297–307. [https://doi.org/10.1016/S1389-9457\(00\)00065-4](https://doi.org/10.1016/S1389-9457(00)00065-4)
- Blanken, T. F., Jansson-Frojmark, M., Sunnhed, R., & Lancee, J. (2021). Symptom-specific effects of cognitive therapy and behavior therapy for insomnia: A network intervention analysis. *Journal of Consulting and Clinical Psychology*, 89(4), 364–370. <https://doi.org/10.1037/ccp0000625>
- Blanken, T. F., Van der Zweerde, T., Van Straten, A., Van Someren, E. J. W., Borsboom, D., & Lancee, J. (2019). Introducing network intervention analysis to investigate sequential, symptom-specific treatment effects: A demonstration in co-occurring insomnia and depression. *Psychotherapy and Psychosomatics*, 88(1), 52–54. <https://doi.org/10.1159/000495045>
- Bootzin, R. R., Epstein, D., & Wood, J. M. (1991). Stimulus control instructions. In P. J. Hauri (Ed.), *Case studies in insomnia* (pp. 19–28). Plenum Press.
- Borsboom, D. (2017). A network theory of mental disorders. *World Psychiatry*, 16(1), 5–13. <https://doi.org/10.1002/wps.20375>
- Carney, C. E., Buysse, D. J., Ancoli-Israel, S., Edinger, J. D., Krystal, A. D., Lichstein, K. L., & Morin, C. M. (2012). The consensus sleep diary: Standardizing prospective sleep self-monitoring. *Sleep*, 35(2), 287–302. <https://doi.org/10.5665/sleep.1642>
- Edinger, J. D., Arnedt, J. T., Bertisch, S. M., Carney, C. E., Harrington, J. J., Lichstein, K. L., ... Martin, J. L. (2021). Behavioral and pharmacological treatments for chronic insomnia disorder in adults: An American Academy of Sleep Medicine systematic review, meta-analysis, and GRADE assessment. *Journal of Clinical Sleep Medicine*, 17(2), 263–298. <https://doi.org/10.5664/jcs.8986>
- Epskamp, S., Borsboom, D., & Fried, E. I. (2018). Estimating psychological networks and their accuracy: A tutorial paper. *Behavior Research Methods*, 50(1), 195–212. <https://doi.org/10.3758/s13428-017-0862-1>
- Epskamp, S., & Fried, E. I. (2018). A tutorial on regularized partial correlation networks. *Psychological Methods*, 23(4), 617–634. <https://doi.org/10.1037/met0000167>
- Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy*, 40, 869–893. [https://doi.org/10.1016/S0005-7967\(01\)00061-4](https://doi.org/10.1016/S0005-7967(01)00061-4)
- Harvey, A. G., Belanger, L., Talbot, L., Eidelman, P., Beaulieu-Bonneau, S., Fortier-Brochu, E., ... Morin, C. M. (2014). Comparative efficacy of behavior therapy, cognitive therapy, and cognitive behavior therapy for chronic insomnia: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, 82(4), 670–683. <https://doi.org/10.1037/a0036606>
- Harvey, A. G., Dong, L., Belanger, L., & Morin, C. M. (2017). Mediators and treatment matching in behavior therapy, cognitive therapy and cognitive behavior therapy for chronic insomnia. *Journal of Consulting and Clinical Psychology*, 85(10), 975–987. <https://doi.org/10.1037/ccp0000244>
- Harvey, A. G., Sharpley, A. L., Ree, M. J., Stinson, K., & Clark, D. M. (2007). An open trial of cognitive therapy for chronic insomnia. *Behaviour Research and Therapy*, 45(10), 2491–2501. <https://doi.org/10.1016/j.brat.2007.04.007>
- Haslbeck, J. M. B., & Waldorp, L. J. (2020). Mgm: Estimating time-varying mixed graphical models in high-dimensional data. *Journal of Statistical Software*, 93(8), 1–46. <https://doi.org/10.18637/jss.v093.i08>
- Isvoranu, A. M., Epskamp, S. (in press). Which estimation method to choose in network psychometrics? Deriving guidelines for applied researchers. *Psychological Methods*. doi: 10.31234/osf.io/mbycn.
- Jansson-Frojmark, M., & Norell-Clarke, A. (2018). The cognitive treatment components and therapies of cognitive behavioral therapy for insomnia: A systematic review. *Sleep Medicine Reviews*, 42, 19–36. <https://doi.org/10.1016/j.smrv.2018.05.001>
- Kazarian, S. S., Howe, M. G., & Csapo, K. G. (1979). Development of the sleep behavior self-rating scale. *Behavior Therapy*, 10, 412–417. [https://doi.org/10.1016/S0005-7894\(79\)80029-5](https://doi.org/10.1016/S0005-7894(79)80029-5), 414.
- Kyle, S. D., Morgan, K., & Espie, C. (2010). Insomnia and health-related quality of life. *Sleep Medicine Reviews*, 14, 69–82. <https://doi.org/10.1016/j.smrv.2009.07.004>
- Maurer, L. F., Espie, C. A., & Kyle, S. D. (2018). How does sleep restriction therapy work? A systematic review of mechanistic evidence and the introduction of the triple-R model. *Sleep Medicine Reviews*, 42, 127–138. <https://doi.org/10.1016/j.smrv.2018.07.005>
- Maurer, L. F., Espie, C. A., Omlin, X., Reid, M. J., Sharman, R., Gavriloff, D., ... Kyle, S. D. (2020). Isolating the role of time in bed restriction in the treatment of insomnia: A randomized, controlled, dismantling trial comparing sleep restriction therapy with time in bed regularization. *Sleep*, 43(11). <https://doi.org/10.1093/sleep/zsaa096>.
- Maurer, L. F., Ftouni, S., Espie, C. A., Bisoudnis, L., & Kyle, S. D. (2021). The acute effects of sleep restriction therapy for insomnia on circadian timing and vigilance. *Journal of Sleep Research*, 30(4), Article e13260. <https://doi.org/10.1111/jsr.13260>
- Morin, C. M. (1993). *Insomnia: Psychological assessment and management*. Guilford press.
- Morin, C. M., & Benca, R. (2012). Chronic insomnia. *Lancet*, 379(9821), 1129–1141. [https://doi.org/10.1016/S0140-6736\(11\)60750-2](https://doi.org/10.1016/S0140-6736(11)60750-2)
- Morin, C. M., Vallières, A., & Ivers, H. (2007). Dysfunctional beliefs and attitudes about sleep (DBAS): Validation of a brief version (DBAS-16). *Sleep*, 30(11), 1547–1554. <https://doi.org/10.1093/sleep/30.11.1547>
- Qaseem, A., Kansagara, D., Forcica, M. A., Cooke, M., Denberg, T. D., & Clinical Guidelines Committee of the American College of P. (2016). Management of chronic insomnia disorder in adults: A clinical practice guideline from the American college

- of physicians. *Annals of Internal Medicine*, 165(2), 125–133. <https://doi.org/10.7326/M15-2175>
- Riemann, D., Baglioni, C., Bassetti, C., Bjorvatn, B., Dolenc Groseelj, L., Ellis, J. G., ... Spiegelhalter, K. (2017). European guideline for the diagnosis and treatment of insomnia. *Journal of Sleep Research*, 26(6), 675–700. <https://doi.org/10.1111/jsr.12594>
- Semler, C. N., & Harvey, A. G. (2004). Monitoring for sleep-related threat: A pilot study of the sleep associated monitoring Index (Sami). *Psychosomatic Medicine*, 66(2), 242–250. <https://doi.org/10.1097/01.psy.0000114870.50968.90>
- Spielman, A. J., Saskin, P., & Thorpy, M. J. (1987). Treatment of chronic insomnia by restriction of time in bed. *Sleep*, 10(1), 45–56.
- van Straten, A., van der Zweerde, T., Kleiboer, A., Cuijpers, P., Morin, C. M., & Lancee, J. (2018). Cognitive and behavioral therapies in the treatment of insomnia: A meta-analysis. *Sleep Medicine Reviews*, 38, 3–16. <https://doi.org/10.1016/j.smrv.2017.02.001>
- Sunnhed, R., Hesser, H., Andersson, G., Carlbring, P., Morin, C. M., Harvey, A. G., & Jansson-Frojmark, M. (2020). Comparing internet-delivered cognitive therapy and behavior therapy with telephone support for insomnia disorder: A randomized controlled trial. *Sleep*, 43(2). <https://doi.org/10.1093/sleep/zsz245>. zsz245.
- Tang, N. K., & Harvey, A. G. (2004). Correcting distorted perception of sleep in insomnia: A novel behavioural experiment? *Behaviour Research and Therapy*, 42(1), 27–39. <https://doi.org/10.1007/s10608-013-9588-2>
- van der Zweerde, T., Bisdounis, L., Kyle, S. D., Lancee, J., & van Straten, A. (2019). Cognitive behavioral therapy for insomnia: A meta-analysis of long-term effects in controlled studies. *Sleep Medicine Reviews*, 48, 101208. <https://doi.org/10.1016/j.smrv.2019.08.002>