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ORIGINAL ARTICLE

'Folk Theories' About the Causes of Insomnia

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Abstract The present study investigates 'folk theories' about the causes of insomnia. Participants with insomnia ($n = 69$) completed a qualitative and quantitative assessment of their folk theories. The qualitative assessment was to speak aloud for 1 min in response to: 'What do you think causes your insomnia?'. The quantitative assessment involved completing the 'Causal Attributions of My Insomnia Questionnaire' (CAM-I), developed for this study. The three most common folk theories for both the causes of one's own insomnia as well as insomnia in others were 'emotions', 'thinking patterns' and 'sleep-related emotions'. Interventions targeting these factors were also perceived as most likely to be viable treatments. Seventy-five percent of the folk theories of insomnia investigated with the CAM-I were rated as more likely to be alleviated by a psychological versus a biological treatment. The results are consistent with research highlighting that folk theories are generally coherent and inform a range of judgments. Future research should focus on congruence of 'folk theories' between treatment providers and patients, and the role of folk theories in treatment choice, engagement, compliance and outcome.

Keywords Insomnia · Folk theories · Causal attributions

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Introduction

The 'folk theories' a patient holds about his/her condition and about treatment may impact treatment choice, engagement, compliance and perhaps outcome. Seligman (1991, 1995) drew attention to the possibility that a patient may believe that a particular treatment would be beneficial and that this belief may affect commitment and adherence to the treatment, therefore contributing to a better outcome. Folk theories may also impact whether patients will seek treatment and the type of treatment they seek.

Cognitive and developmental psychologists have long been interested in 'folk theories' (e.g., Carey 1985; Gopnik and Meltzoff 1997; Wellman and Gelman 1988). Like scientific theories, folk theories involve interrelated sets of beliefs about the domain in question, with an emphasis on the causal and explanatory relationships between entities in that domain (Lombrozo 2012). Folk theories are generally coherent and inform a range of judgments. For example, Kim and Ahn (2002b) found that folk theories held by clinicians about mental illness and the causal relationships between symptoms and diagnostic criteria determine which symptoms are considered most important or "central" to the illness (see also Kim and Ahn 2002a; Murphy and Medin 1985 for other examples of coherence). In a series of three experiments, Ahn et al. (2009) showed that mental

health clinicians' conceptualize mental disorders along a single continuum with 'biological disorders' at one end and 'non-biological disorders' at the other and that these beliefs informed judgments about effective treatments. The clinicians believed that medication would be more effective for 'biological' disorders and psychotherapy more effective for 'psychosocially'-based disorders. Characterizing folk theories is of interest not only because it provides insight into the structure of human cognition, but also because folk theories mediate behavior. This has been suggested in the context of 'folk theories' of how a thermostat works (which can determine how one sets a thermostat at home; Kempton 1986) and compellingly demonstrated for 'folk theories' of intelligence (which has an impact on school performance and how one responds to feedback; Dweck 2000).

In the context of patients with a mental illness, the study of folk theories has been limited to the literature on treatment preference. Although there have been some mixed results (Glass et al. 2001; King et al. 2005), a recent meta-analysis by Swift and Callahan (2009) of 26 studies and 2,300 patients indicated that patients who are matched for their treatment preference and the treatment they received are more likely to improve and less likely to drop out compared to those who are mismatched. Other studies suggest that type of treatment and outcome measured may be important. Specifically, patients with depression were randomized to supportive-expressive psychotherapy, sertraline or placebo (Iacoviello et al. 2007). Therapeutic alliance, the relationship between the patient and therapist, is a robust predictor of treatment success and was the chosen outcome. Patients preferring psychotherapy and who received psychotherapy experienced increases in their therapeutic alliance over time, whereas those who received sertraline or placebo experienced decreases in therapeutic alliance over time. There were no differences for patients who preferred medication. However, in two more recent studies also comparing psychotherapy and medications for depression, but with different outcome measures, a mismatch between the preferred and actual treatment was associated with lower remission, higher depression (Kocsis et al. 2009), greater attrition, poorer attendance and less positive working alliance (Kwan et al. 2010), for *both* psychotherapy and medications.

The target disorder for this study is insomnia, which is a prevalent problem, reported by approximately 10–25 % of the population (Hajak et al. 2011). The consequences of insomnia are severe and include functional impairment, work absenteeism, impaired concentration and memory, increased use of medical services and increased risk of accident, health problems and the development of psychiatric disorders. Not surprisingly given the prevalence and associated impairments, the cost to society is enormous (Daley et al. 2009; Hajak et al. 2011). The present study sought to assess the 'folk theories' patients with insomnia hold about their insomnia and how this relates to engagement with treatment and outcome. We already know that patients with insomnia believe that cognitive arousal is the main determinant of their insomnia, relative to somatic arousal (Lichstein and Rosenthal 1980). We also know that patients with insomnia are more likely to prefer a psychological treatment over a biological treatment (Morin et al. 1992; Vincent and Lionberg 2001). On the other hand, Ahn et al.'s (2009) study of clinicians' beliefs suggests that when it comes to sleep disorders, biological explanations dominate both psychological and environmental explanations. We seek to extend this knowledge by examining a broader range of possible folk theories in a diagnosed treatment-seeking patient group, to establish the extent to which folk theories are associated with beliefs about the effectiveness of a psychological treatment versus a biological treatment, to assess whether individuals consider the causes of their insomnia to be different from those for others with their disorder, and to determine whether these beliefs influence the efficacy of treatment.

Within the context of an NIMH-funded treatment trial for chronic insomnia, the present study was designed to address four aims. The first aim was to assess the folk theories held by patients with insomnia about the causes of their own insomnia and insomnia in other people, and to establish the relationship between these. Based on previous research, we predicted that thinking patterns (i.e., cognitive arousal) would be the most common folk theory, relative to somatic arousal. We also predicted that folk theories of one's own insomnia would be positively correlated with folk theories of insomnia in others. The second aim had two parts. We sought to assess folk theories held by insomnia patients about the likely efficacy of various treatment targets for their own insomnia and insomnia in other people, and to establish the relationship between these. We also sought to establish whether these beliefs link to the individuals' folk theories of the causes of their insomnia. Based on previous research indicating coherence between folk theories and related beliefs, we predicted that patients who endorsed a particular cause for insomnia would cite interventions targeting that cause as a viable treatment. Aim three was to assess folk theories about 'psychological treatments' versus 'biological treatments'. On the one hand, prior research on patients with insomnia suggests that psychological explanations will be endorsed more readily than biological explanations, especially among participants who were interested in receiving a psychological treatment. On the other hand, research on clinicians' beliefs about the causal bases of DSM-IV-TR disorders suggests that when it comes to sleep disorders, biological explanations predominate both psychological and environmental explanations (Ahn et al. 2009). The

fourth aim was to begin to devise a method for measuring patient folk theories about mental illness.

Method

Participants

Participants were 69 adults (ages 25–65) with chronic insomnia recruited to participate in an NIMH-funded treatment trial for chronic insomnia. Participants were recruited through internet advertisements and flyers distributed to psychiatric clinics in the community. A telephone interview was completed to screen for eligibility. Individuals who were considered likely to be eligible based on the initial telephone screen were invited to the clinic for an extensive clinical evaluation.

The inclusion criteria for this study were: (a) adults at least 25 years old, (b) reported difficulties initiating and/or maintaining sleep, defined as a sleep onset latency and/or wake after sleep onset $\geq$30 min, with a corresponding sleep time $\leq$6.5 h per night, as ascertained by daily sleep diaries kept for a 1-week baseline period, and presence of insomnia for a minimum of 3 nights per week, (c) reported that the sleep disturbances (or associated daytime fatigue) caused significant distress or impairment in social, occupational, or other areas of functioning as measured by a rating of at least 2 on items 5 or 7 on the Insomnia Severity Index, and (d) reported that the difficulties had been present for a minimum of 6 months.

The exclusion criteria were (a) presence of an active, progressive or unstable physical illness (e.g., congestive-heart failure, cancer, COPD, acute pain) or neurological degenerative disease (e.g., dementia, multiple sclerosis) directly related to the onset of insomnia, (b) use of medications known to alter sleep (e.g., steroids, theophylline, propanolol), (c) lifetime diagnosis of any psychotic disorder, (d) individuals at risk of suicide, (e) alcohol or drug abuse within the past year, (f) current or past psychological treatment of insomnia (CBT; at least 2 appointments) within the past 5 years, (g) using alcohol as a sleep aid, any alcohol after 7:00 p.m. or more than two alcoholic beverages per day, with participants required to discontinue all of these practices at least 2 weeks prior to baseline assessment, (h) pregnant woman or breast-feeding mother, and (i) individuals using more than four caffeinated beverages per day, with participants required to reduce their caffeine intake below that level for the duration of the study or be excluded from the study and (j) possible sleep apnea, restless legs or periodic limb movements during sleep, or a circadian-based sleep disorder were excluded via the DUKE interview.

Treatment data is not presented as 27 participants did not begin treatment; while they met the inclusion/exclusion criteria for the current study, they did not meet criteria for the stricter inclusion criteria for the NIMH treatment trial.

At the first visit, 0 % of patients were taking mood stabilizers, 2.9 % antidepressants, 0 % antipsychotics, 1.4 % anxiolytics, and 17.4 % prescription sleep aids.

Measures

Participants' diagnoses were assessed using the Structured Clinical Interview for DSM-IV (SCID; First et al. 1995) and the Duke Structured Interview for Sleep Disorders (DSISD; Edinger et al. 2004). The SCID is a semi-structured interview designed to assess DSM-IV diagnostic criteria for Axis-I disorders. The SCID has shown good reliability for the majority of disorders it covers (Skre et al. 1991; Williams et al. 1992). The DSISD is a semi-structured interview that assesses research diagnostic criteria for sleep disorders. Various questionnaires were administered including the Insomnia Severity Index (ISI; Bastien et al. 2001), which was administered as a measure of the severity of insomnia. The ISI has established psychometric properties (Bastien et al. 2001; Morin et al. 2011).

In the absence of a psychometrically validated alternative, the 'Causal Attributions of My Insomnia Questionnaire' (CAM-I) was developed specifically for this study. The construction of the questionnaire involved three steps. First, we conducted a detailed survey of the existing literature and existing measures for possible beliefs about the causes of insomnia. Second, the investigators met on several occasions to devise a comprehensive list. Third, to ensure there were no ceiling/floor effects, no salient additional factors and no serious confusions on the part of participants, the materials were pre-tested on 20 participants without insomnia.

The CAM-I is a self-report measure that presents 12 domains that 'may contribute to your insomnia.' Each domain included a clarification. 'Sleep-Related Thoughts' was clarified as 'thinking about falling asleep or consequences of not sleeping', 'Hormonal Factors' as 'aging, menstrual cycle changes, menopause', 'Bodily Arousal' as 'heart racing, jittery, nervous feelings', 'Genetic Factors' as 'runs in family, genetic anomaly', 'Lifestyle Factors' as 'diet, exercise', 'Thinking Patterns' as 'can't shut off thoughts, reviewing events from the day', 'Biochemical Factors' as 'chemical imbalance, neurotransmitter levels', 'Environmental Factors' as 'loud noises, partner snoring, children, pets', 'Scheduling' as 'irregular sleep and wake times', 'Sleep-related Emotions' as 'stress, anxiety about falling asleep or consequences of not sleeping', 'Emotions' as 'stress, anxiety, excitement about events, work, or family', and 'Developmental Factors' as 'childhood experiences,

traumatic events'. The questionnaire asks participants to rate the likelihood that: (Qu1) these factors contribute to *your* insomnia, (Qu2) these factors contribute to insomnia *in other people*, (Qu3) *your* insomnia could be alleviated by a treatment targeting these factors, (Qu4) *other people's* insomnia could be alleviated by a treatment targeting these factors, (Qu5) a psychological treatment (e.g., therapy) targeting these factors could help alleviate *your* insomnia, and (Qu6) a biological treatment (e.g., medication) targeting these factors could help alleviate *your* insomnia. The 12 domains were presented in a random order for each participant. In addition, the ordering of the last two questions was counterbalanced. Likert-type ratings were made on a 7-point continuum with three anchors: 'Very likely;' 'Neither likely nor unlikely;' and 'Very unlikely.' The CAM-I was administered via pen and paper.

Procedure

The data reported in this paper were collected in the context of an NIMH-funded randomized controlled trial of three cognitive behavioral treatments (CBT) for chronic insomnia. All procedures were approved by the University of California, Berkeley, Committee for the Protection of Human Subjects. All assessments were completed by trained and carefully supervised postdoctoral and doctoral student interviewers.

Given that it is expensive and time consuming to recruit patient samples, combining a qualitative and quantitative approach within this study seemed judicious. Following written informed consent, the 'speak aloud' procedure was administered (Halford et al. 2002; Harvey et al. 2008), where the patient was asked 'What do you think causes your insomnia?'. If the participant stopped talking before 1 min elapsed, the assessor prompted 'Can you say more about that?'. During piloting we were surprised that patients were exceptionally fast at describing the causes of their insomnia. One minute was ample. Next the participant completed a questionnaire packet that included a sociodemographics form, the ISI and the 'Causal Attributions of My Insomnia Questionnaire' (CAM-I). The assessor then collected the participants' sleep diaries and administered the DSISD, the SCID and several other interviews not relevant to the current study.

Data Analysis

The 1-min description of 'What do you think causes your insomnia' was recorded and was later carefully transcribed verbatim and then divided into utterance units, defined as a clause containing only one thought, action or idea (Harvey and Bryant 1999). Two independent raters, blind to group status, coded each script for the causes of insomnia. There was 97 % inter-rater agreement. Disagreements were resolved via discussion.

Results

Demographic and Sleep Characteristics

Table 1 presents the sample demographic and sleep characteristics. Participants were more likely to be male (57.6 %), Caucasian (69.6 %) and non-Hispanic (92.8 %). The average Insomnia Severity Index score ($M = 18.4$; $SD = 3.65$) was in the moderate 'clinical' range and average total sleep time and sleep efficiency were 5.5 h and 71 %, respectively.

Table 1 Sociodemographic and sleep characteristics

	N	%
Gender		
Male	40	58.0
Female	29	42.0
Race		
White	48	69.6
Asian	7	10.1
Black	6	8.7
Other	5	7.2
Not specified	3	4.3
Ethnicity		
Hispanic	4	5.8
Non-hispanic	64	92.8
Not specified	1	1.4
	M	SD
Age (years)	46.62	10.80
Insomnia severity index	18.41	3.65
Sleep over past week (via sleep diary)		
Bed time	11:26 pm	51.61 min
Wake time	6:38 am	68.79 min
Arising time	7:23 am	64.60 min
Sleep onset latency (min)	42.77	41.57
Number of nighttime awakenings	2.08	1.21
Wake after sleep onset (min)	53.95	35.70
Early morning awakening (min)	45.56	42.52
Total sleep time (min)	334.92	69.06
Time in bed (min)	477.45	63.64
Sleep efficiency (%)	70.86	14.80
Sleep quality[a]	2.16	0.64

M mean, *SD* standard deviation

[a] Sleep quality was on a scale of 1 (very bad) to 5 (very good)

Aim 1: Folk Theories of Insomnia: Patient Perspectives

Fifty-four participants (78 % of total sample) provided audible narratives. The total number of causes coded was 180, and the average number of causes reported was 2.98 (SD = 1.64). The categories broadly match the categories already included in the CAM-I, with notable exceptions listed in the third column of Table 2.

As is evident in Table 3, of the 12 factors rated in the CAM-I, Emotions, Thinking Patterns and Sleep-Related Emotions were most commonly endorsed as factors that 'contribute to your insomnia' (Qu1). A repeated measures ANOVA comparing responses to Question 1 across the 12 factors was significant, $F(1,68) = 10.23$, $p < .001$. Paired t tests ($p < .01$ to control for multiple comparisons) were conducted to assess for differences in specific factors. Emotions, Thinking Patterns, Sleep-Related Emotions and Sleep-Related Thoughts were the most highly rated categories.

Of the 12 factors rated, Emotions, Thinking Patterns and Sleep-Related Emotions were also most commonly endorsed as factors that 'contribute to insomnia in other people' (Qu2 in Table 3). A repeated-measures ANOVA comparing responses to Question 2 across the 12 factors was significant, $F(1,68) = 9.80$, $p < .001$. Paired t tests ($p < .01$ to control for multiple comparisons) were conducted to assess for differences in specific factors. Emotions, Sleep-Related Emotions and Thinking Patterns were most highly rated.

Significant medium-to-large correlations between the answer to 'How likely do you think it is that these factors contribute to your insomnia?' (Qu1) and 'How likely do you think it is that these factors contribute to insomnia in other people?' (Qu2) were observed (see Table 3). Across all 12 factors, ratings for Qu2 are higher than for Qu1, $t(68) = -1.64$ to -7.01, $p < .003$.

Table 2 Qualitative coding of 'what do you think causes your insomnia?'

Cause	*N*	CAM-I category	Proposed additional CAM-I category
Thinking patterns	29	Thinking patterns	
Stress	28		Stress
Don't know	20		Don't know
Emotions	15	Emotions	
Hard to relax	14	Bodily arousal	
Sleeping patterns	12	Scheduling	
Environment	9	Environmental factors	
Diet	9	Lifestyle factors	
Lack of exercise	8	Lifestyle factors	
Genes	7	Genetic factors	
Caffeine	5	Lifestyle factors	
Pain/uncomfortable	6		Body sensations
Trauma	5	Developmental factors	
Hormones	4	Hormonal factors	
Sleep architecture abnormalities	4	Biochemical factors	
Parenting/childhood	3	Developmental	
Anticipation	2	Emotions	
Developmental factors	2	Developmental factors	
Hypervigilance	2	Bodily arousal	
Relationships/marriage problems	2		Interpersonal problems
Alertness	1	Bodily arousal	
Clock watching	1	Environmental factors	
Go to bathroom	1		Body sensations
Light sleepers	1	Bodily arousal	
Medications	1	Biochemical factors	
Not enough time alone	1	Environmental factors	
Self-worth	1		Self-concept

Aim 2: Folk Theories About Treatment Targets and Relationship to Folk Theories About Causes

Thinking patterns, emotions and sleep-related emotions were also the most highly rated for the likelihood that 'your insomnia could be alleviated by targeting these factors' (Qu3; see Table 3). A repeated measures ANOVA comparing responses to Question 3 across the 12 factors was significant, $F(1,68) = 14.30$, $p < .001$. Paired t tests ($p < .01$ to control for multiple comparisons) were conducted. As evident in Table 3, thinking patterns, emotions, sleep-related emotions, sleep-related thoughts were rated as more credible treatment targets for one's own insomnia.

Thinking patterns, emotions and sleep-related emotions were also the most highly rated for the likelihood that 'other people's insomnia could be alleviated by targeting these factors' (Qu4; see Table 3). A repeated measures ANOVA comparing responses to Question 4 across the 12 factors was significant, $F(1,68) = 10.14$, $p < .001$. Paired t tests ($p < .01$ to control for multiple comparisons) were conducted. As evident in Table 3, emotions, sleep-related emotions and thinking patterns were rated as more credible treatment targets for other people's insomnia.

Significant medium-to-large correlations between the answer to 'How likely do you think it is that your insomnia could be alleviated by a treatment targeting these factors?' (Qu3) and 'How likely do you think it is that other people's insomnia could be alleviated by a treatment targeting these

Table 3 Folk theories of the causes for *your* and *other people's* insomnia

Contributing factor	Qu1		Qu2		Qus 1/2	Qu3		Qu4		Qus 3/4	Qus 1/3	% 'Likely'* response to Q1 who believe a treatment targeting this factor would be effective
	M	SD	*M*	SD	*r*	*M*	SD	*M*	SD	*r*	*r*	
Sleep-related thoughts$_{st}$	4.99	1.81	5.71	1.10	0.46*	5.10	1.70	5.64	1.11	0.47*	0.90*	95.6 %
	st > ba,g,d		*st > g*			*st > g,en,d*		*st > g*				
Hormonal factors$_{h}$	4.35	2.13	5.54	1.22	0.56*	4.07	1.79	5.23	1.32	0.64*	0.75*	76.3 %
			h > g					*h > g*				
Bodily arousal$_{ba}$	4.01	2.32	5.54	1.21	0.64*	4.18	2.12	5.47	1.17	0.57*	0.88*	91.2 %
			ba > g					*ba > g*				
Genetic factors$_{g}$	4.19	2.02	4.88	1.33	0.54*	4.17	1.79	4.71	1.44	0.71*	0.73*	83.3 %
Lifestyle factors$_{l}$	4.51	2.02	5.65	1.28	0.57*	4.59	1.96	5.59	1.26	0.50*	0.83*	89.5 %
	l > d		*l > g*					*l > g*				
Thinking patterns$_{t}$	5.78	1.58	6.19	0.97	0.36*	6.00	1.25	6.04	1.05	0.60*	0.74*	96.6 %
	t > st,h,ba,g,l,bc,en,sc,d		*t > st,h,ba,g,l,bc,en,d*			*t > st,h,ba,g,l,bc,en,sc,d*		*t > st,h,ba,g,l,bc,en,d*				
Biochemical factors$_{bc}$	4.55	1.90	5.41	1.24	0.35*	4.69	1.72	5.41	1.24	0.47*	0.76*	88.9 %
	bc > d					*bc > sc,l*		*bc > g*				
Environmental factors$_{en}$	4.39	2.24	5.51	1.29	0.39*	4.12	2.11	5.29	1.44	0.35*	0.86*	71.8 %
			en > g									
Scheduling$_{sc}$	4.57	2.07	5.78	1.22	0.48*	4.63	2.01	5.72	1.17	0.47*	0.93*	92.7 %
	sc > d		*sc > g*					*sc > h,g*				
Sleep related emotions$_{se}$	5.41	1.77	5.96	1.10	0.63*	5.57	1.56	5.85	1.12	0.62*	0.85*	94.3 %
	se > ba,g,l,en,sc,d		*se > st,h,ba,g,bc,en,d*			*se > st,h,ba,g,l,bc,en,sc,d*		*se > h,ba,g,bc,en*				
Emotions$_{em}$	5.97	1.61	6.23	1.02	0.57*	5.96	1.38	6.16	1.02	0.60*	0.88*	93.3 %
	em > st,h,ba,g,l,bc,en,sc,se,d		*em > h,ba,g,l,bc,en,sc,d*			*em > se,h,ba,g,l,bc,en,sc,d*		*em > st,h,ba,g,l,b,se,sc,d*				
Developmental factors$_{d}$	3.62	2.31	5.43	1.27	0.57*	3.64	2.31	5.46	1.35	0.50*	0.94*	100 %
	l > d		*d > g*					*d > g*				

r = Pearson correlation. Q1 = How likely do you think it is that these factors contribute to *your* insomnia? Q2 = How likely do you think it is that these factors contribute to insomnia in *other people*? Q3 = How likely do you think it is that *your* insomnia could be alleviated by targeting these factors? Q4 = How likely do you think it is that *other people's* insomnia could be alleviated by targeting these factors? Q1-4 = rated on a 7-point continuum with three anchors: 1 = 'Very Unlikely', 4 = 'Neither Likely nor Unlikely;' and 7 = 'Very Likely.' *'Likely' rating was a rating of 5, 6, or 7 on Question 1. Subscripts document significant differences ($p < .01$) between factor means that are indicated with corresponding factor abbreviations following each factor name

M mean, *SD* standard deviation

*** $p < .001$

Table 4 Folk theories of the treatment for insomnia

Contributing factor	Qu5: How likely do you think it is that a psychological treatment targeting these factors could help alleviate *your* insomnia?[a]		Qu6: How likely do you think it is that a biological treatment targeting these factors could help alleviate *other people's* insomnia?[a]		
	M	SD	*M*	SD	*t*
Sleep-related thoughts	5.22	1.67	4.13	1.93	5.44***
Hormonal factors	3.93	2.11	4.01	2.08	−0.33
Bodily arousal	4.16	2.59	3.83	2.14	2.80**
Genetic factors	4.10	1.96	4.03	1.88	0.36
Lifestyle factors	4.59	1.95	3.91	2.07	3.51**
Thinking patterns	5.87	1.21	4.59	1.87	5.95***
Biochemical factors	4.75	1.61	4.84	1.62	−0.52
Environmental factors	4.09	2.07	3.43	2.00	2.91**
Scheduling	4.52	2.38	3.72	2.49	4.16***
Sleep-related emotions	5.41	2.36	3.96	2.97	5.80***
Emotions	5.72	2.26	4.65	1.98	5.55***
Developmental factors	4.17	2.24	3.28	2.17	4.15***

M mean, *SD* standard deviation

[a] Questions 5 and 6 were rated on a 7-point continuum with three anchors: 'Very Unlikely;' 'Neither Likely nor Unlikely;' and 'Very Likely'

* $p < .05$; ** $p < .01$; *** $p < .001$

factors?' (Qu4) were observed (see Table 3). Thus, if a participant rated treatment targets as plausible for themselves, they generally found the treatment target plausible for others. Significant medium-to-large correlations between the answer to 'How likely do you think it is that these factors contribute to your insomnia' (Qu1) and 'How likely do you think a treatment targeting these factors would help your insomnia' (Qu3) were observed (see Table 3). In other words, factors contributing to insomnia were also considered plausible treatment targets. Among patients who gave a "likely" rating (5, 6 or 7) to 'How likely do you think it is that these factors contribute to your insomnia' (Qu1), the proportion of patients who believed that targeting this factor in treatment could alleviate their insomnia was above 70 % for all possible contributors (see Table 3).

Aim 3: Perceived Effectiveness of Biological Versus Psychological Treatments

A paired-samples *t* test comparing the average of ratings of 'How likely do you think it is that a psychological treatment targeting these factors could help alleviate your insomnia?' (Qu5) and 'How likely do you think it is that a biological treatment targeting these factors could help alleviate your insomnia?' (Qu6) indicated that psychological treatments were rated as more likely to alleviate the insomnia, $t(68) = 4.43$, $p < .001$ (Psychological $M = 4.71$, $SD = 1.16$; Biological $M = 4.03$, $SD = 1.40$). As reported in Table 4, the ratings for a psychological treatment were highest for Thinking Patterns, Emotions and Sleep-Related Thoughts. The ratings for a biological treatment were highest for Thinking Patterns, Biochemical Factors and Emotions. A psychological treatment was rated as significantly more likely than a biological treatment to alleviate 9 of the 12 (74 %) contributors; namely, Sleep-Related Thoughts, Bodily Arousal, Lifestyle Factors, Thinking Patterns, Environmental Factors, Scheduling, Sleep-Related Emotions, Emotions and Developmental Factors.

Discussion

The first aim was to assess the folk theories held by patients with insomnia about the causes of their own insomnia and insomnia in other people. The three most common folk theories cited for both the cause of one's own insomnia as well as insomnia in others were Emotions, Thinking Patterns and Sleep-Related Emotions. The importance of 'thinking patterns', the CAM-I phrasing for cognitive arousal, is consistent with our prediction and previous research (e.g., Lichstein and Rosenthal 1980). The importance of emotions (both sleep-related and other emotions) underscores growing recognition of the importance of the emotional level of explanation across disorders (Kring 2009; Kring and Werner 2004), and in insomnia specifically (Baglioni et al. 2010; Harvey et al. 2009). We also sought to establish the relationship between the folk theories held by patients with insomnia about the causes of their

own insomnia and insomnia in other people. Consistent with our prediction, folk theories of the causes of one's own insomnia were uniformly positively correlated with folk theories of insomnia in others (r's range 0.35–0.64 in Table 3). This finding is consistent with evidence from the cognitive psychology literature showing that folk theories tend to be coherent and inform a range of judgments (e.g., Kim and Ahn 2002a, b), although this study is the first to investigate the relationship between folk theories of the self and other in a clinical context. Interestingly, across all 12 factors ratings for Qu2 are higher than for Qu1, $t(68) = -1.64$ to -7.01, $p < .003$, meaning that the participants rated each factor as more likely to explain other people's insomnia than their own. These findings make sense in light of prior research. On the one hand, people treat themselves as an anchor for drawing inferences about other people (e.g., Epley et al. 2004; Nickerson 1999). On the other hand, and consistent with the current result, research in social psychology indicates that people tend to think they are more exceptional and unique than is likely warranted (e.g., Goethals et al. 1991; Kruger 1999).

The second aim sought to assess folk theories held by insomnia patients about the likely efficacy of various treatment targets for their own insomnia and insomnia in other people. The three causes rated as the most viable treatment targets for one's own insomnia, as well as insomnia in others, were 'thinking patterns', 'emotions' and 'sleep related emotions'. In other words, the same factors that were most likely to be perceived to be causes were also the factors most likely to be perceived as viable targets for treatment. Notably, the relationship between ratings of likelihood that one's own insomnia could be alleviated by targeting each factor were consistently positively correlated with the likelihood that other people's insomnia could be alleviated by targeting each factor (r's range 0.35–0.71 in Table 3) and between 71.8 and 100 % of patients who responded that a factor was a 'likely' cause of their insomnia also believed that a treatment targeting that factor would be effective, again pointing to coherence between folk theories and related belief domains (e.g., Kim and Ahn 2002a, b).

The third aim assessed folk theories about 'psychological treatments' versus 'biological treatments'. Consistent with the prior research on patients with insomnia suggesting that psychological treatments will be endorsed more readily than biological treatments (Morin et al. 1992; Vincent and Lionberg 2001), especially among participants who were applying to receive a psychological treatment, 9 of the 12 causes were rated as more likely to be alleviated by a psychological versus a biological treatment. This is contrary to previous research suggesting that clinicians' favor biological explanations when it comes to DSM-IV-TR sleep diagnoses (Ahn et al. 2009). Future research may identify which disorders patients and treatment providers agree versus disagree about when it comes to their folk theories, and how this impacts treatment provision, choice, engagement, compliance and outcome. As we expected, participants did not identify clearly biological factors—hormones, genetic factors, and biochemical factors—as being more amenable to treatment with a psychological intervention versus a biological treatment.

The qualitative analysis pointed to the potential to improve the CAM-I by refining the existing categories and potentially formulating new categories. Participants' responses to the open-ended interview question largely fit within the current categories, which suggests that the initial factors were relatively comprehensive. However, several folk theories generated by participants did not fit in the current categories or cross-cut multiple categories, such as stress, interpersonal problems, body sensations and self-concept. We suggest that body sensations is distinct from bodily arousal, with the former capturing pain, discomfort and the need to visit the bathroom and the latter referring to anxiety-related sensations such as tenseness, increased heart rate and an 'on edge' feeling (Lichstein and Rosenthal 1980). This evidence provides a basis for the development of new factors.

There are several limitations and domains for future research. First, this was a relatively small treatment-seeking sample of individuals with chronic insomnia. Folk theories may differ for patients who do not seek treatment. Future research should seek to recruit a larger sample so that psychometrics can be established. Second, the study included individuals seeking a psychological treatment for insomnia and excluded individuals who experienced medical conditions that caused or exacerbated their insomnia. This may have reduced the number of participants who endorsed folk theories that were more biological in nature. Interestingly, only 24.6 % of patients indicated that they had previously tried a prescribed medication. This reduces the concern that the sample is biased by a previous attempt at a biological treatment that was minimally effective. Third, there are several variables that might influence attributions within insomnia samples that could be explored in future research. Rarely for an insomnia sample, our sample included more men than women. Hence, we took the opportunity to assess gender differences on the major variables. The only difference was that women were more likely to believe that biological treatments would alleviate their insomnia ($M = 4.24$; SD = 0.88), relative to men ($M = 3.76$; SD = 1.50) ($t = -1.22$; $p < .05$). Perhaps there would also be difference for patients with a childhood-onset versus adulthood-onset (Espie et al. 2012). In the present sample, one patient reported childhood onset and five reported adolescent onset. The remainder of the sample reported onset in adulthood. So while the sample

size for child/adolescent onset was too small to explore this variable in the present study, this may be a fruitful domain for future research.

In conclusion, consistent with previous research, thinking patterns (i.e., cognitive arousal) were prominent in patients' folk theories of insomnia, as were emotional factors. Coherence between folk theories about the causes of one's own insomnia, theories about the causes of others' insomnia and the factors that are viable targets for treatment was consistently observed. Together these findings are the first steps for future studies focused on the extent to which patient folk theories impact treatment choice, engagement, compliance and outcome, with the potential for efforts in the development of treatments that target changing folk theories when there is a mismatch between the folk theory and the treatment of choice.

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Conflict of Interest Dr. Morin serves on consulting/advisory boards for Merck, Valeant, Purdue, Novartis, Eli Lilly and the speaker's bureau for Valeant and Merck. The other authors have no conflict of interest disclosures.

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