INSOMNIA

TREATMENT OPTIONS
TREATMENT EFFECTIVENESS

HOW DO WE TX THIS ?

A HX PERSPECTIVE

PHARMACOTHERAPY

QUEEN'S HOSPITAL, BIRMINGHAM.

CASES UNDER THE CARE OF DR. SAWYER.

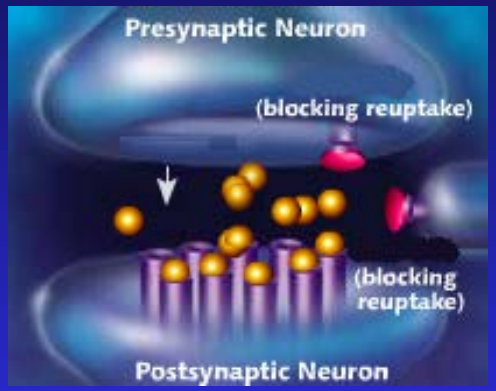
Insomnia.— is usually successfully treated by full doses of bromides conjoined with tincture of ergot and cod-liver oil. If the insomnia be serious, it must be stopped at once by hypnotics, preferably by opium.

PAST AND CURRENT THERAPEUTIC APPROACH TO PHARMACOTHERAPY

TREATMENT OPTIONS

CLASSIC THERAPIES

- Benzodiazepines (e.g., temazepam)
- Imidazopyridines (e.g., zolpidem)
  Pyrazolopyrimidine (e.g., zaleplon)
  Pyrrolopyrazine (e.g., eszopiclone)



NEWER THERAPIES

- Doxepin (e.g., “silenor)
- Melatonin Agonists (e.g., ramelteon)
- Orexin antagonists (e.g., suvorexant)

OFF LABEL

- Antidepressants (e.g., amitriptyline, trazodone)
- Antipsychotics (e.g., quetiapine)

IN DEVELOPMENT / COMBO

- 2nd Generation DORAs
- BZRAs + CBT-I
- Stimulants + CBT-I

Table 1
Basic pharmacology of medications used to treat insomnia

Agent	Trade name	Class	FDA indication	t_{max} (h)	$t_{1/2}$ (h)	Binding profile							Metabolism
						Benzo binding	Anti-H1	MT1–MT2	Anti-5HT2	Anti-alpha-1	Anti-dopa	Anti-mAch	
Flurazepam	Dalmane	Benzodiazepine	Insomnia	0.5–1.5	40–250	+++							CYP2C19, CYP3A4
Quazepam	Doral	Benzodiazepine	Insomnia	2	20–120	+++							CYP3A4, CYP2C19
Estazolam	Prosom	Benzodiazepine	Insomnia	1.5–2	10–24	+++							CYP3A4
Temazepam	Restoril	Benzodiazepine	Insomnia	1–3	8–20	+++							Glucuronide conjugation
Triazolam	Halcion	Benzodiazepine	Insomnia	1–3	2–5.5	+++							CYP3A4, glucuronide conjugation
Clonazepam	Klonopin	Benzodiazepine	Seizures, anxiety	1–2	35–40	+++							CYP2B, CYP3A4, acetylation
Lorazepam	Ativan	Benzodiazepine	Anxiety	1–3	12–15	+++							Glucuronide conjugation
Alprazolam	Xan												/5, CYP2C19
Diazepam	Vali												CYP2C19, CYP3A4, nide conjugation
Chlordiazepoxide	Libr												CYP2C19, CYP3A4, nide conjugation
Zolpidem (MR)	Amb												, CYP1A2, CYP2C9
Zaleplon	Son												le oxidase, CYP3A4
Eszopiclone	Lun												, CYP2E1
Ramelteon	Roz												, CYP2C, CYP3A4
Amitriptyline	Elav												, CYP2C19, CYP2D6,
Doxepin	Sinequan	Tertiary amine tricyclic	MDD, anxiety	1.5–4	10–30	+++				+++		+	CYP3A4, CYP2C19, CYP2D6, CYP2C9, CYP1A2
Trazodone	Desyrel	Chlorophenylpiperazine	MDD	1–2	7–15				+++	+++			CYP3A4, CYP2D6, CYP1A2
Mirtazapine	Remeron	Tetracyclic	MDD	0.25–2	20–40		+++		+++				CYP2D6, CYP1A2, CYP3A4
Quetiapine	Seroquel	Dibenzothiazepine	Schizophrenia mania	1	7		++		+	+++	+		CYP2D6, CYP3A4
Olanzapine	Zyprexa	Thiobenzodiazepine	Schizophrenia mania	5	30		+++		+++	++	++	+++	CYP1A2
Risperidone	Risperdal	Benzisoxazole	Schizophrenia mania	1	3–20		+		+++	+++	++		CYP2D6, CYP3A4
Diphenhydramine	Benadryl	Ethanolamine	Allergy, OTC sleep aid	2–3	5–11		+++					+++	CYP2D6, CYP1A2, CYP2C9, CYP2C19
Doxylamine succinate	Unisom	Ethanolamine	Allergy, OTC sleep aid	1.5–2.5	10–12		+++					+++	CYP2D6, CYP1A2, CYP2C9

MDD = major depressive disorder; $t_{1/2}$ includes the half-lives of the parent compound and major active metabolites; OTC = over-the-counter; information in table from Refs. [9–53]. Anti-5HT2-Serotonin Type 2 Receptor Antagonist; Anti-alpha-1 - Alpha 1 Adrenergic Antagonist; Anti-Dopa - Dopamine Antagonist; Anti-H1 - Antihistamine; Anti-mAch - Muscarinic Cholinergic Antagonist; Benzo – Benzodiazepine; CYP - Cytochrome P450; ETOH – Alcohol; MR – Modified release; MT1-MT2 - Melatonin Type 1 and Type 2 Receptor Antagonist; $t_{1/2}$ - Half-life; t_{max} - Time to maximum blood level.

WHILE COMPREHENSIVE,
WHAT IS MISSING FROM THIS TABLE ?

Krystal Sleep Medicine Reviews 13 (2009) 265–274

Drugs Indicated for Insomnia

Generic	Brand	$T_{1/2}$ (Hours)	Dose (mg)	Drug Class
Flurazepam	Dalmane	48-120	15-30	BZD
Temazepam	Restoril	8-20	15-30	BZD
Triazolam	Halcion	2-6	0.125-0.25	BZD
Estazolam	Prosom	8-24	1-2	BZD
Quazepam	Doral	48-120	7.5-15	BZD
Zolpidem	Ambien	1.5-2.4	5-10	non-BZD
Zaleplon	Sonata	1	5-20	non-BZD
Eszopiclone	Lunesta	5-7	1-3	non-BZD
Zolpidem Ext. Rel.	Ambien CR	1.5-2.4*	6.25-12.5	non-BZD
Ramelteon	Rozerem	1.5-5	8	MT agonist

Compiled by Dan Buysse

PLUSES & MINUSES FOR EACH TREATMENT MODALITY

SPEND TIME HERE ON CONCEPTS

Benzodiazepines (e.g., Temazepam)

+ Good short term efficacy
+ Low interaction profile (vs. predecessors)
+ Relatively High LD (vs. predecessors)
+ Minor side effects (depending on 1/2 life)

- Not recommended for long term use
- Not curative (gains are lost when Tx is d/c ?)
- Rebound insomnia
- Suppresses SWS or REM
- Abuse Potential (?)
- Dependence (?) ASIDE: ANXIETY AND/OR PAIN

PLUSES & MINUSES FOR EACH TREATMENT MODALITY

Imidazopyridines / Non-benzodiazepines (e.g., Zolpidem, Zaleplon, Zopiclone)

+ Good “short” term efficacy
+ May be used safely up to 6 months (FDA SI REMOVED)
+ Low interaction profile
+ High LD
+ Few side effects
+ Doesn’t suppress SWS or REM
+ Does not result in rebound insomnia

- Not curative (gains are lost when Tx is d/c)
- Parasomnogenesis (pegged to zolpidem)
- Abuse Potential (?)

PLUS & MINUSES FOR EACH TREATMENT MODALITY

Low Dose Tricyclics – Doxepin (not silenor)

+ Good short term efficacy (WASO only)
+ Good durability (3 months)
+ No appreciable effects on Sleep Architecture
+ Minor side effects at hypnotic doses (?)
+ Data exists for long term administration in MDD
+ Low abuse potential

- Interacts with other meds (?)
- Possible cardiovascular effects (?)
- Anticholinergic side effects (?)
- Not curative (gains are lost when Tx is d/c)

PLUSES & MINUSES FOR EACH TREATMENT MODALITY

Melatonin Agonists (M1 & M2 receptor agonists)

Ramelteon (Rozerem)

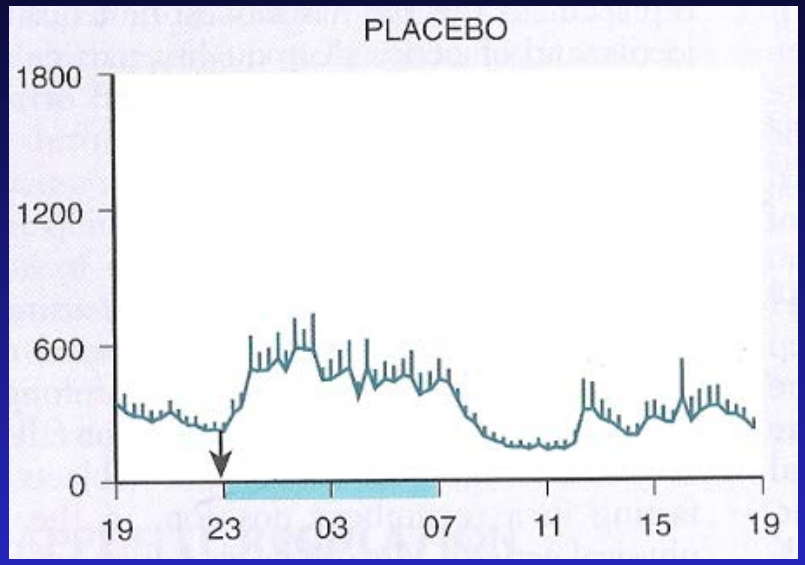
PLUSES & MINUSES FOR EACH TREATMENT MODALITY

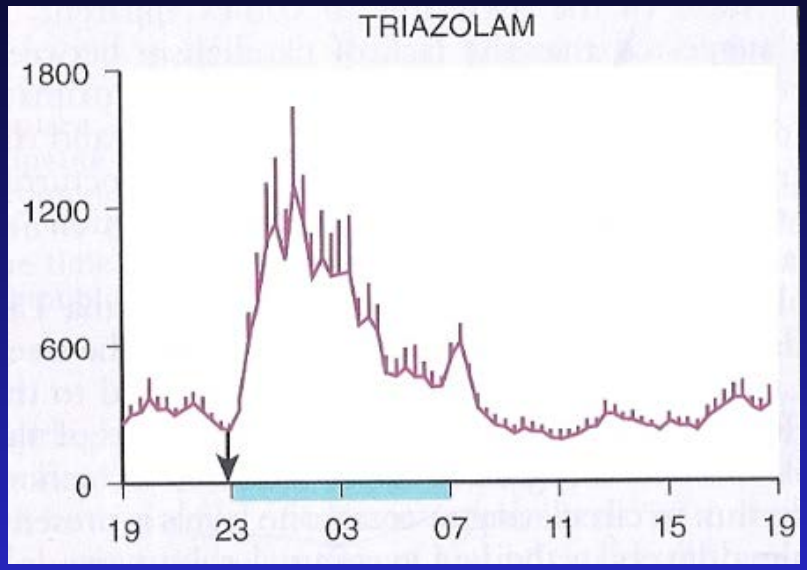
Melatonin Agonists (M1 receptor agonists)

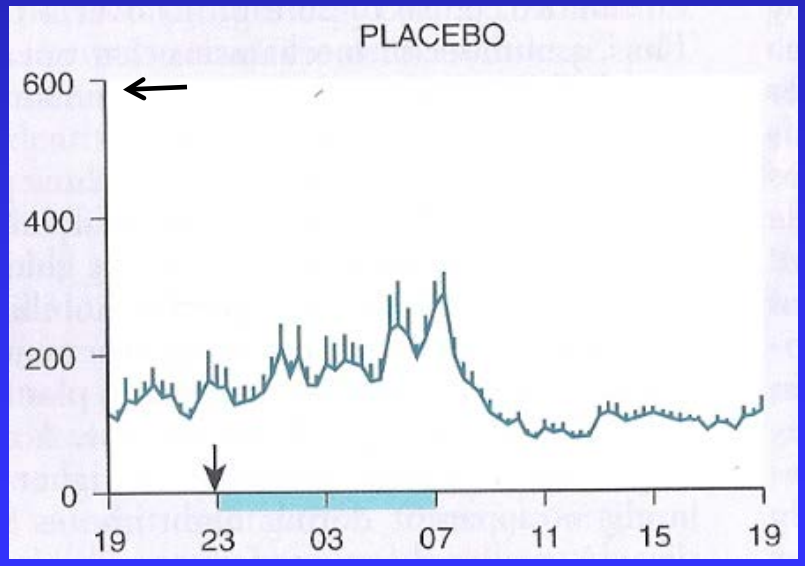
+ “Established” efficacy
+ May be used safely for extended intervals
+ Low interaction profile (except fluvaxamine)
+ High LD
+ Few side effects (?)
+ Doesn’t suppress SWS or REM
+ Does not result in rebound insomnia

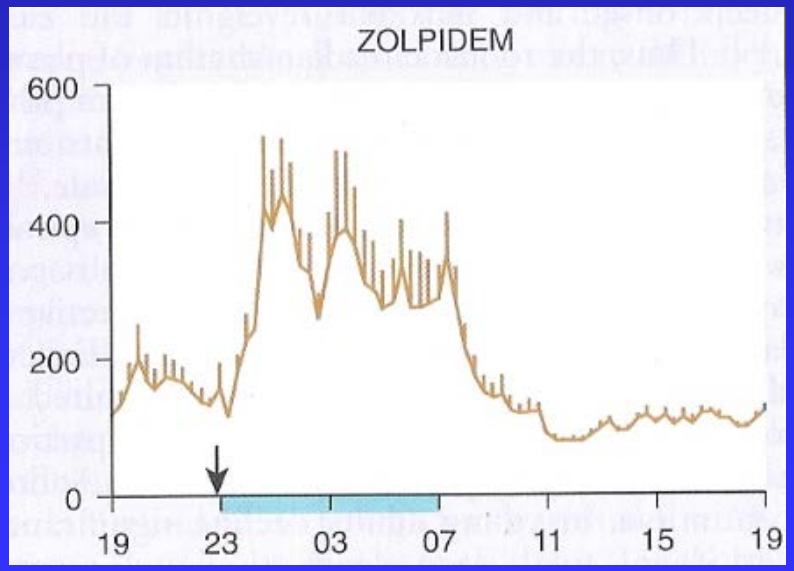
- Not curative (gains are lost when Tx is d/c)
- May have reproductive hormone effects (hyperprolactinemia)

24 HOUR PROLACTIN SECRETION









PPSM 5TH EDITION – VAN CAUTER

PLUSES & MINUSES FOR EACH TREATMENT MODALITY

Orexin Antagonists (DORAs)

Belsomra (Suvorexant)

PLUSES & MINUSES FOR EACH TREATMENT MODALITY

Orexin Antagonists (DORAs)

Almorexant / Suvorexant

+ Established efficacy
+ Low interaction profile
+ High LD
+ Few side effects
+ Doesn’t suppress SWS or REM
+ Does not result in rebound insomnia

- Not curative (gains are lost when Tx is d/c)
- Parasomnogensis (in label)
- Weakness or Sleep Paralysis (in label)
- Narcoleptogenesis ??
- May not be safe for pregnant women

PLUS & MINUSES FOR EACH TREATMENT MODALITY

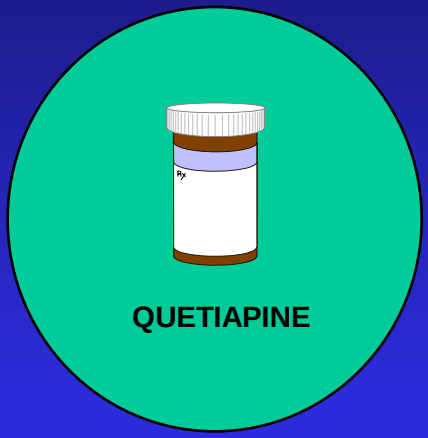
Antidepressants (e.g., Amitriptyline, Trazodone)

+ Good short term efficacy (?)
+ Minor side effects at hypnotic doses (?)
+ Data exists for long term administration in MDD
+ Low abuse potential

- Interact with other meds (?)
- Possible cardiac toxicity (?)
- Anticholinergic side effects (?)
- PLMs as an iatrogenic effect (more so w/ amitriptyline)
- Off label prescription for Primary Insomnia
- Not curative (gains are lost when Tx is d/c)
- Rebound insomnia (?)
- Suppresses REM (not so much trazodone)
- Priapism

WHAT ABOUT ANTIPSYCHOTICS ?

WHAT ABOUT QUETIAPINE ?



Psychopharmacology (2008) 196:337–338
DOI 10.1007/s00213-007-0968-8

LETTER TO THE EDITORS

Quetiapine in primary insomnia: a pilot study

Michael H. Wiegand · Florentina Landry · Torsten Brückner · Corina Pohl · Zdenko Veselý · Thomas Jahn

Received: 21 September 2007 / Accepted: 26 September 2007 / Published online: 6 October 2007

In the treatment of primary insomnia, benzodiazepines and non-benzodiazepine hypnotics are not a first-line medication, mainly due to the dependency risk, especially in long-term treatment. Sedating antidepressants are widely used as an alternative treatment (Wiegand 1999). However, these compounds have several side effects and tend to interact with other drugs, which is a problem especially in elderly patients.

For these reasons, neuroleptics play an increasing role in the management of chronic insomnia. In clinical practice, the use of neuroleptics in acute and chronic insomnia is traditionally widespread, e.g., melperone, pipamperone, promethazine, etc., especially in elderly insomniacs, as a sleeping medication. However, this is based on unsystematic clinical experience rather than on data: Systematic studies on the efficacy of neuroleptics in this indication are extremely limited. This is true for older compounds as well as for the more advanced and in many respects advantageous compounds like risperidone, olanzapine, and quetiapine.

Among the newer, "atypical" antipsychotics, quetiapine appears to be a specially promising "candidate" for the treatment of chronic insomnia in general and primary insomnia in particular. This dibenzothiazepin derivate has antagonistic effects on several central neurotransmitter systems: serotonin ($5\text{-}HT_{1A}$ and $5\text{-}HT_2$), dopamine (D_1 and D_2), histamine (H_1), and on adrenergic α_1 and α_2 receptors. There is virtually no action on cholinergic, muscarinergic, and benzodiazepine receptors. This special profile suggests a favorable effect on sleep, especially the combination of a $5\text{-}HT_2$ receptor and an H_1 receptor blockade. Some antidepressants have a similar profile, e.g., mirtazapine, trazodone, and trimipramine. All of these compounds are used in the treatment of chronic insomnia (unrelated to depression).

Quetiapine's favorable effect on sleep in healthy probands as well as in depressed patients has been demonstrated by an increasing number of studies (e.g., Wetter et al. 2002; Cohrs et al. 2004; Calabrese et al. 2005; Sokolski and Brown 2006; Todder et al. 2006; Baune et al. 2007; Endicott et al. 2007.)

In an open pilot study, we treated 18 outpatients suffering from primary insomnia with quetiapine for 6 weeks. After a washout period of 1 week, medication was given at bedtime. The dose was 25 mg initially; in seven patients, it was increased to 50 mg, in one patient, to 75 mg. Sleep parameters were measured by repeated polysomnographies. Subjective sleep quality was assessed by means of the Pittsburgh Sleep Quality Index (PSQI; Buysse et al. 1989) and sleep diaries. Cognitive effects were assessed by means of a neuropsychological test battery.

The results demonstrated that quetiapine in a comparably low dose improved objective and subjective sleep parameters in patients with primary insomnia (Table 1). This improvement was already present after 2 weeks' medication, and then increased after 6 weeks' medication. The improvement was most clear in the subjective sleep variables as assessed by means of the PSQI and the patients' sleep diaries. The

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Table 1 Selected objective and subjective sleep parameters resulting from polysomnography and PSQI ratings

	T_1 (baseline)	T_2 (2 weeks med.)	p	T_3 (6 weeks med.)	p
Objective sleep quality (polysomnography)					
Subjective sleep quality (PSQI scores)					
Total score	13.1±2.3	9.1±3.3	0.00	6.8±3.3	0.00

Presented are means±SD. "*p*" refers to the change from baseline (Wilcoxon's test, two-tailed).
REM Rapid eye movements, *SPT* sleep period time, *PSQI* Pittsburgh Sleep Quality Inventory

WHAT ABOUT PROSPECTIVE SAMPLING DATA

NAME: _____ DATE _____

COMPLETE IMMEDIATELY BEFORE BED CONCERNING HOW YOU FELT TODAY:

	MON	TUES	WED	THUR	FRI	SAT	SUN		MEAN
TYPICAL DAY? (YES/NO) *									
FATIGUE (NONE 0—1—2—3—4—5 A LOT)									
STRESS (NONE 0—1—2—3—4—5 A LOT)									
ALERT (NOT VERY 0—1—2—3—4—5 VERY)									
CONCENTRATION (GOOD 0—1—2—3—4—5 BAD)									
MOOD (BAD 0—1—2—3—4—5 GOOD)									
TIME SPENT EXERCISING (MIN.)									
TIME SPENT OUTSIDE TODAY (MIN.)									
NUMBER OF ALCOHOLIC BEVERAGES									
PRESCRIPTIONS TODAY (YES/NO)									
OTC MEDS TODAY (YES/NO)									
PAIN TODAY (NONE 0—1—2—3—4—5 A LOT)									
HEALTH (FELT FINE 0—1—2—3—4—5 BAD)									
MENSTRUATE TODAY (YES/NO)									
MENSTRUAL PAIN (NONE 0—1—2—3—4—5 BAD)									

** PLEASE INDICATE ON THE BACK OF THIS SHEET WHY ANY GIVEN DAY WAS NOT TYPICAL AND/OR WHAT MEDICATIONS YOU TOOK ON ANY GIVEN DAY.

COMPLETE IMMEDIATELY ON AWAKENING

	MON	TUES	WED	THURS	FRI	SAT	SUN		MEAN
TIME TO BED (CLOCK TIME)									
TIME OUT OF BED (CLOCK TIME)									
TIME TO BED (DEV FRM 11)									
TIME OUT OF BED (DEV FRM 7)									
(SL) TIME TO FALL ASLEEP									
(NUMA) NUMBER TIMES AWAKENED									
(WASO) WAKE AFTER SLEEP ONSET									
(TTOB) TOTAL AMOUNT TIME OUT OF BED									
(TST) TOTAL SLEEP TIME (MIN.)									
SLEEP QUALITY (GOOD 0—1—2—3—4—5 POOR)									
FATIGUE (NONE 0—1—2—3—4—5 A LOT)									

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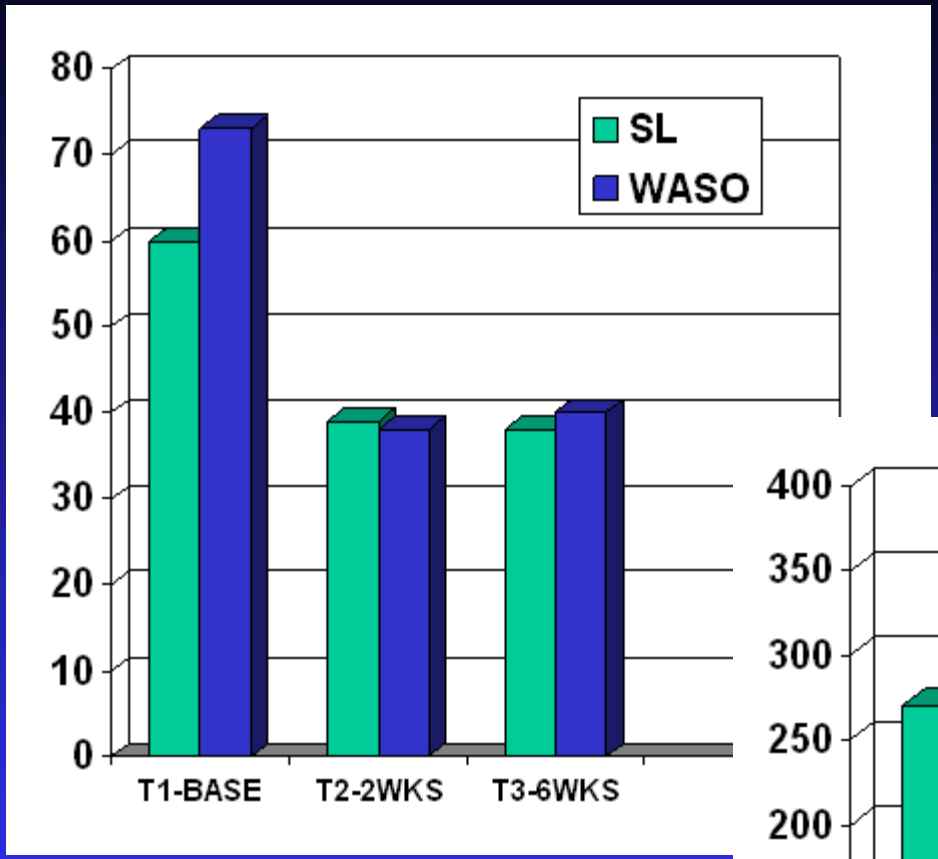
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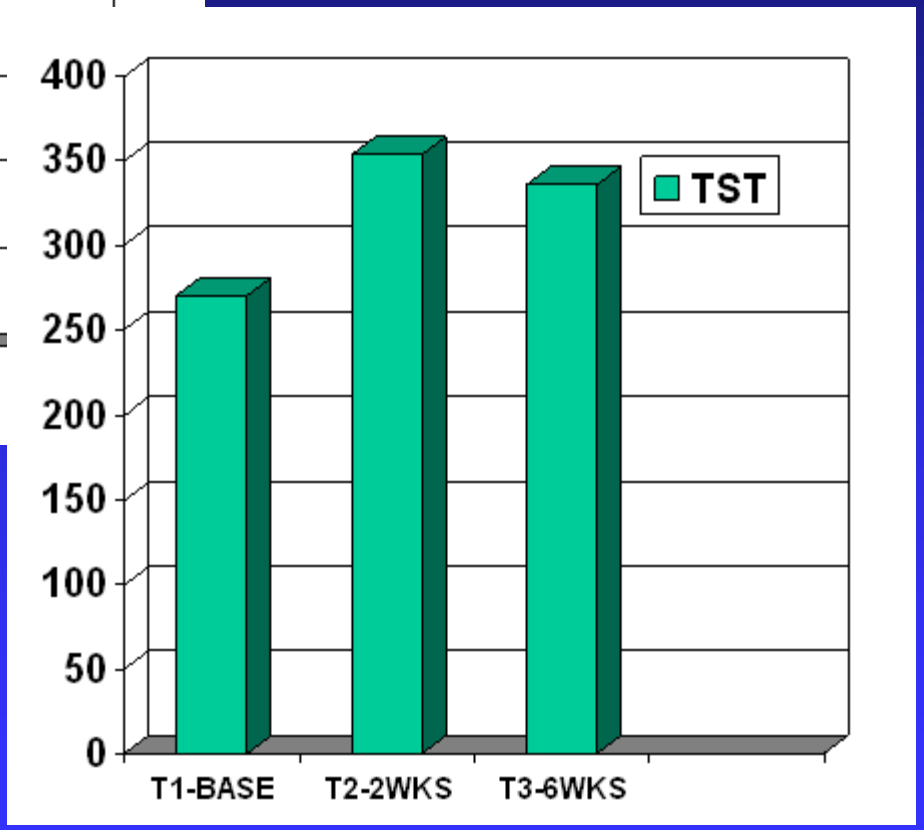
T3-6WKS


400
350
300
250
200
150
100
50
0
TST
T1-BASE
T2-2WKS
T3-6WKS

Quetiapine for Primary Insomnia: A Double Blind, Randomized Controlled Trial

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Objective: *To evaluate the clinical efficacy of Quetiapine 25 mg for the treatment of primary insomnia.*
Material and Method: *A randomized, double-blind, placebo-controlled clinical trial was conducted. Patients with DSM-IV-TR defined primary insomnia were asked to record a sleep diary one week prior to treatment, followed by 2 weeks of nightly treatment with either Quetiapine 25 mg or placebo. The primary outcomes were total sleep time (TST), sleep latency (SL), daytime alertness and functioning and sleep satisfaction; side effects were recorded as secondary outcome. Data were collected between January 2007 and December 2007, at Srinagarind Hospital of Khon Kaen University.*
Results: *Thirteen patients completed the present study (mean age 45.95 years old; range 25-62). Quetiapine group increased mean TST by 124.92 minutes and 72.24 minutes in the placebo group. Mean SL was reduced by 96.16 minutes in the Quetiapine group and 23.72 minutes in the placebo group. Statistical significance was not reached between both groups. In the Quetiapine group two patients reported side effects of dry lips, dry tongue and morning drowsiness.*
Conclusion: *The present study is the first study to evaluate the effect of Quetiapine in primary insomnia in a randomized controlled trial. Quetiapine at 25 mg at night did show a trend for improvement of TST and reduced SL in primary insomnia with few side effects but not reaching statistical significance. A study with a larger sample size is needed to demonstrate its efficacy.*

Keywords: *Insomnia, Primary Insomnia, Quetiapine, Sleep*

J Med Assoc Thai 2010; 93 (6): 729-34
Full text. e-Journal: *http://www.mat.or.th/journal*

Insomnia is among the most common concerns in clinical practice[1]. Numbers of studies have assessed the prevalence of insomnia. The findings vary, depending mostly on the criteria used for insomnia and the population studied. Data from the National Institutes of Health Epidemiologic Catchment Area (ECA) study, approximately 10-15% of adults in the United States suffer from chronic insomnia and 25-35% has transient or occasional insomnia[2,3]. In European countries insomnia occurred in 19% of the population[4]. Only one study from Thailand reported the prevalence of 46.3% in elderly[5].

From the Diagnostic and Statistical Manual of Mental Disorders, Fourth edition Text Revision (DSM-IV-TR) criteria, primary insomnia is characterized by the predominant complaint of difficulty initiating or maintaining sleep, or of non-restorative sleep, for at least 1 month. This sleep disturbance should not be caused by another diagnosable sleep disorder or mental disorder[6]. Primary insomnia accounts for 12-15% of patients with chronic insomnia. The essential features are conditioned arousal in response to efforts to sleep and negative expectations about sleep[7]. Because insomnia is a subjective complaint, polysomnography is the only method that provides a comprehensive measurement of sleep but it is expensive and unpractical. The use of subjective assessment methods such as self report questionnaires and sleep logs are more often used in clinical studies[8,9].

Primary insomnia treatments include both pharmacological and non-pharmacological modalities. Pharmacotherapies aim to reduce morbidity and prevent complications. Medication should be given for short-term management. Benzodiazepines and sedating antidepressants are commonly used.

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BOTTOM LINE

WHILE THE SUBJECTIVE EFFICACY DATA APPEAR SOLID, THE JUDGEMENT (IN THE ABSENCE OF COMPARATIVE EFFICACY AND SAFETY DATA) APPEARS TO BE THAT RISKS OF THIS APPROACH OUTWEIGH ITS POTENTIAL BENEFITS

IN SUM

BZRAs HAVE GOOD EFFICACY
AND APPEAR REASONABLE SAFE

SADs APPEAR TO HAVE GOOD EFFICACY
THERE ARE CONCERNS ABOUT SIDE EFFECTS

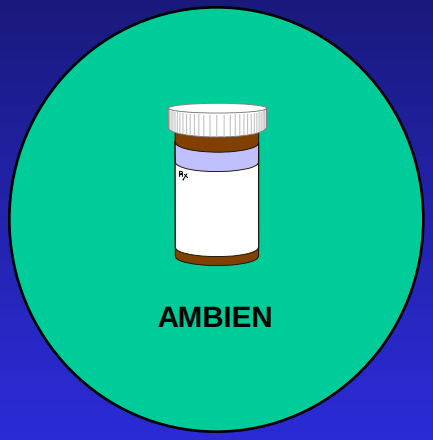
MELATONIN AGONISTS ARE “IFFY”
THERE ARE CONCERNS ABOUT SIDE EFFECTS

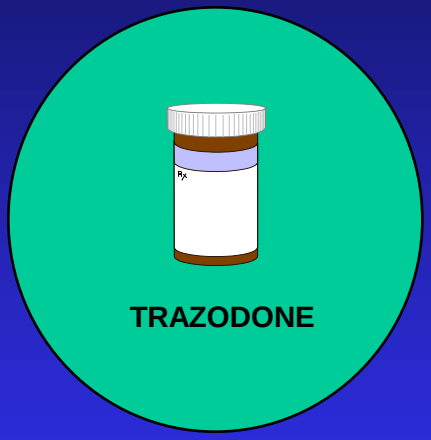
SUVOREXANT SHOWS GOOD OBJECTIVE GAINS
THERE ARE CONCERNS ABOUT SIDE EFFECTS

ANTIPSYCHOTICS
HAVE TOO NARROW A RISK BENEFIT RATIO

COMPARATIVE EFFICACY

HOW DO HYPNOTICS COMPARE WITH SEDATING ANTIDEPRESSANTS ?





I would have guessed …

TRAZODONE AND ZOLPIDEM TREATMENT OF PRIMARY INSOMNIA

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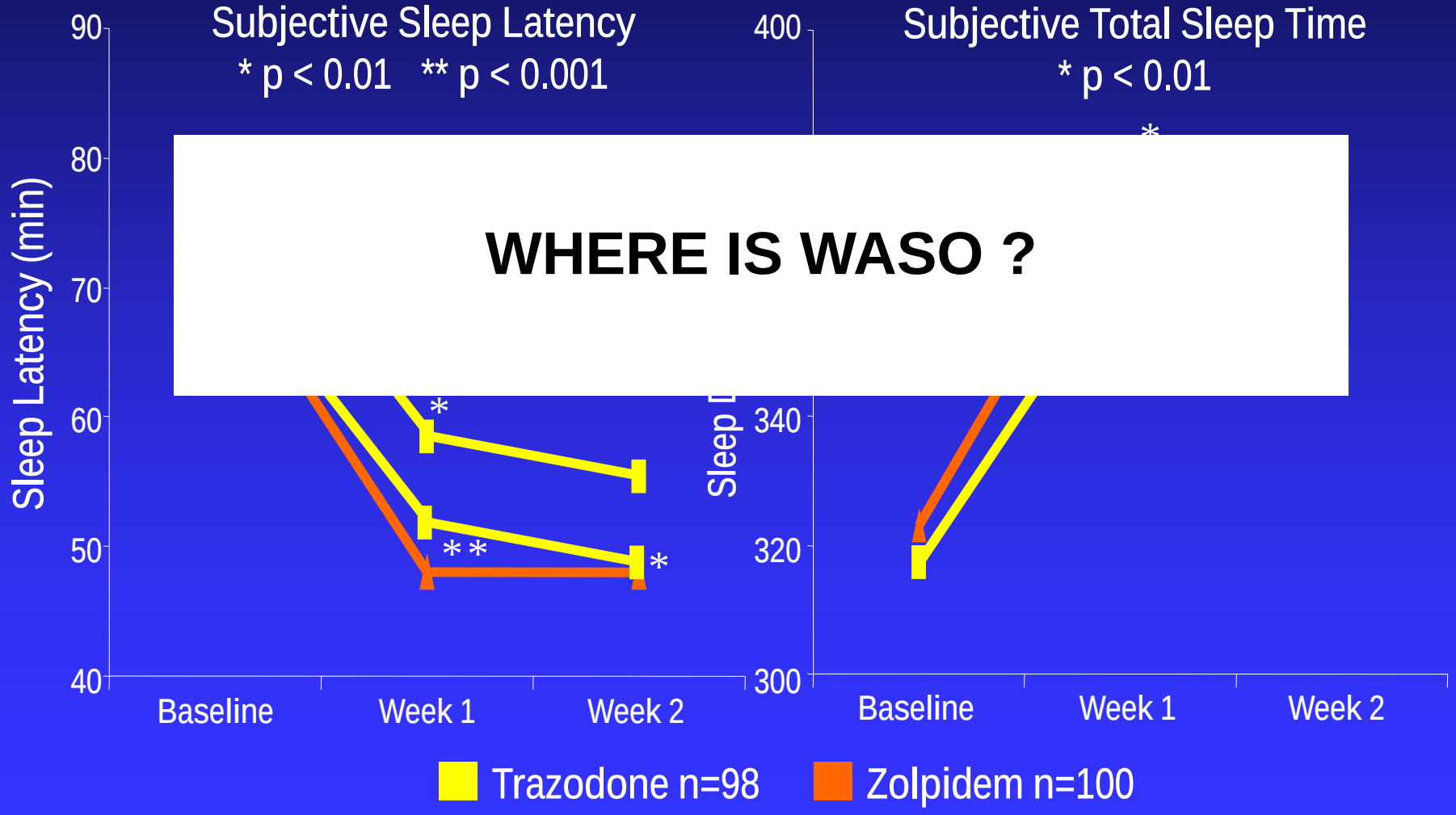
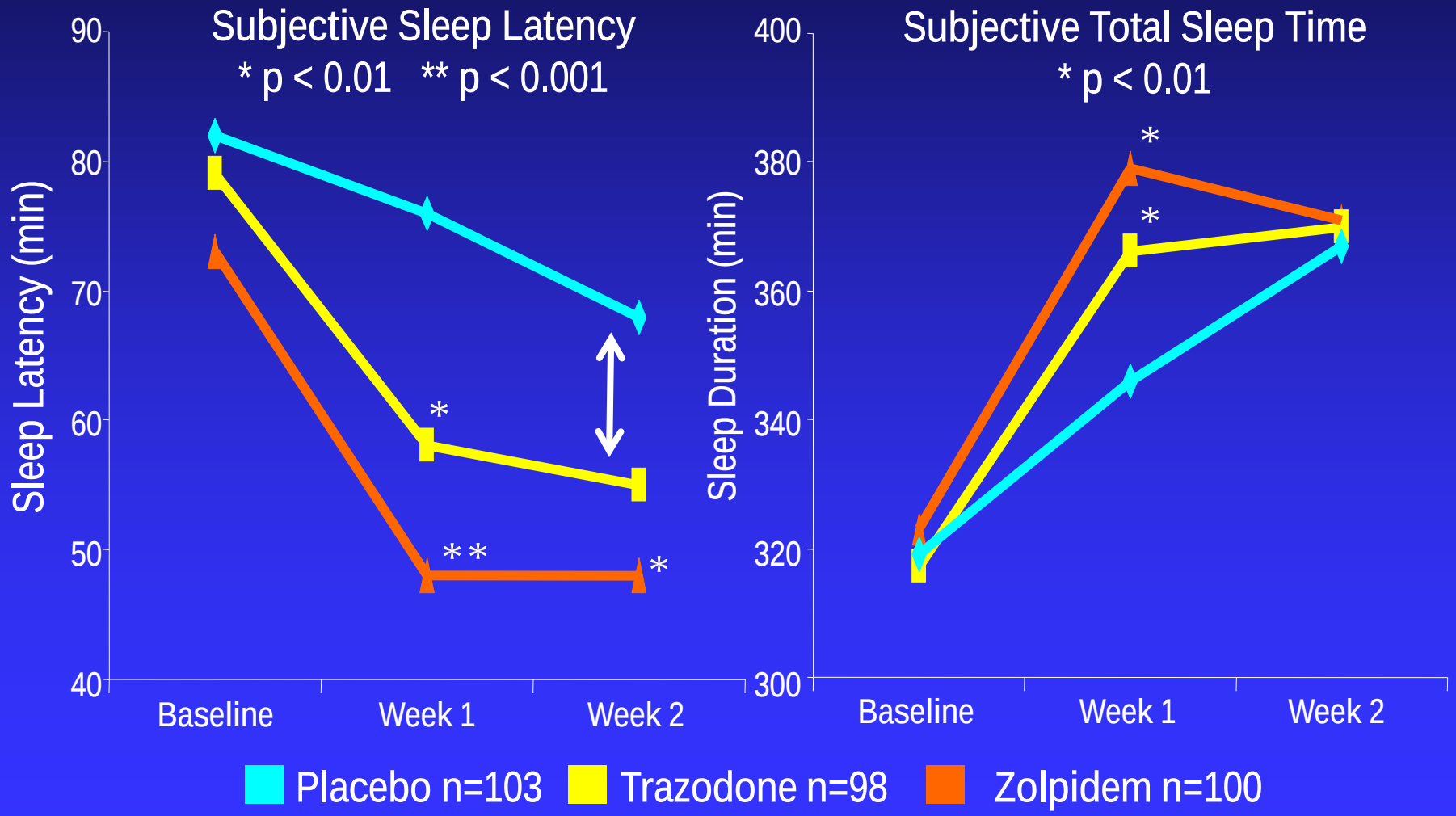


WHERE IS WASO ?

TRAZODONE AND ZOLPIDEM TREATMENT OF PRIMARY INSOMNIA

Walsh, *Hum Psychopharmacol*, 1998



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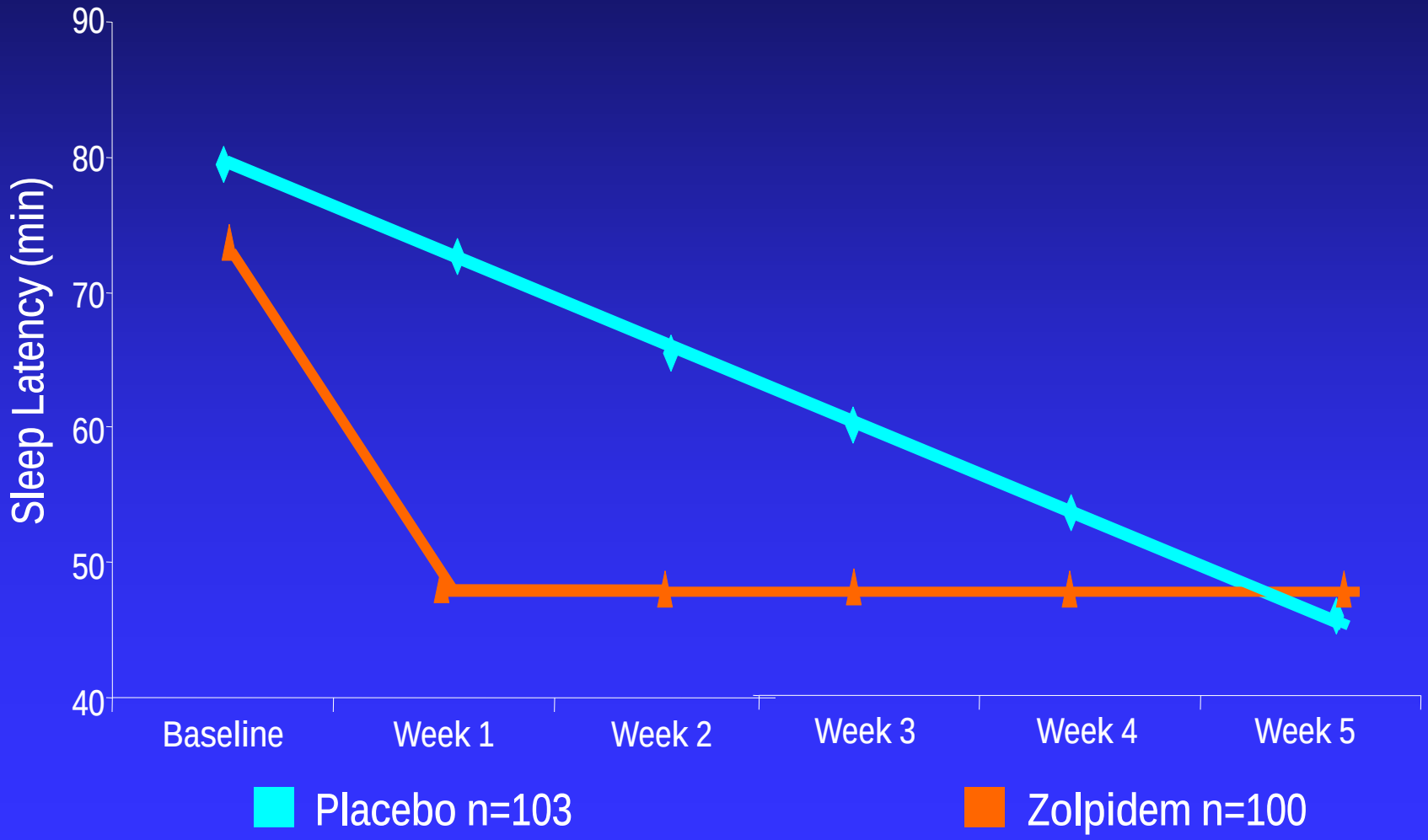

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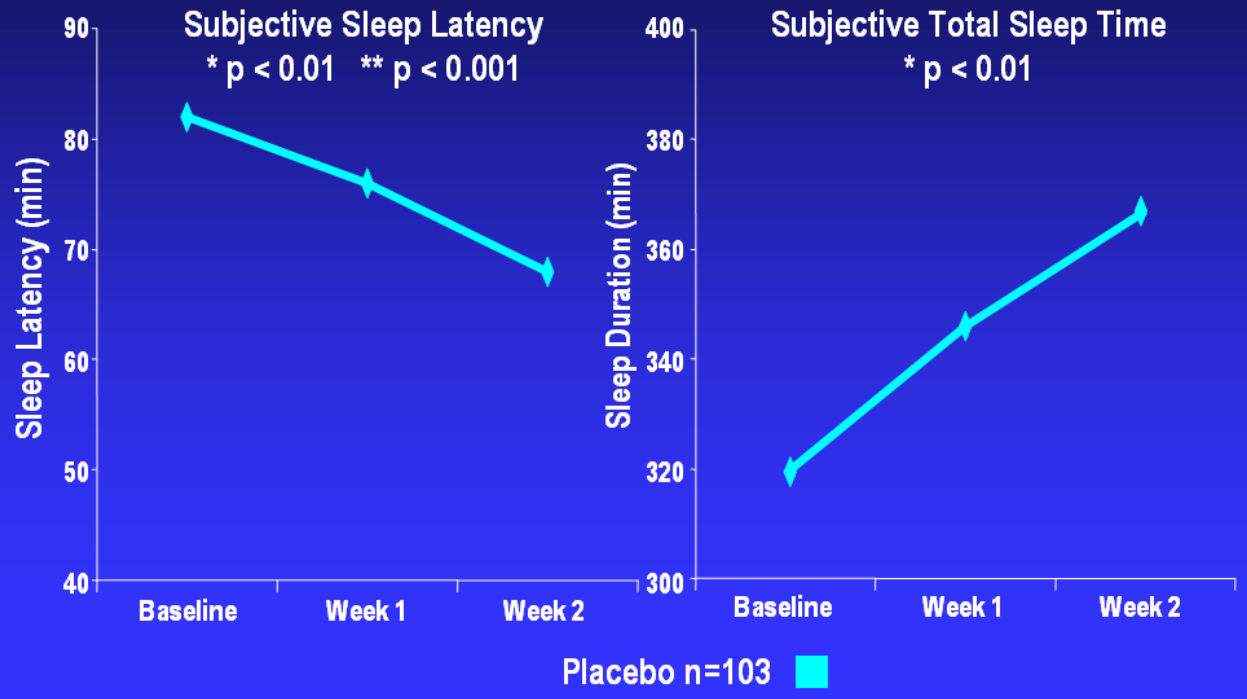

“What’s up with placebos and insomnia ?!!”

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THEORETICAL REVIEW

Placebo effects in primary insomnia

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KEYWORDS
Insomnia;
Randomized Clinical trials;
Placebo effects;
Periodicity of insomnia

Summary Placebo effects are commonly observed in insomnia clinical trials. With the advent of longer-term trials, such effects appear to be remarkably robust and durable. In this paper we review the classic factors that are believed to contribute to placebo effects and how these factors operate in insomnia randomized clinical trials. Beyond this we suggest that the episodic nature of insomnia may interact with patient preferences for intermittent dosing in such a way as to sustain placebo effects in the long term. An appreciation of the latter phenomenon may provide increased power to detect therapeutic outcomes and may be used to potentiate clinical gains.

Introduction

It is a common finding within insomnia randomized clinical trials (RCTs) that placebos produce significant changes on self reported sleep continuity measures.[1] In a recent meta-analysis of such effects,[1] McCall and colleagues estimated the magnitude of pre to post change on sleep latency and total sleep time measures to be approximately 20%. Longer-term trials (both intermittent and nightly dosing) show that such effects are not only stable but that clinical improvements continue to occur over time. A representation of placebo effects for several recent trials is contained in Fig. 1.

The purpose of the present article is to review - the traditional explanations for what the placebo effect is and to advance a hypothesis that placebo effects may be maintained over long periods of time as a result of a peculiar interaction between illness severity, pill taking behavior, and interval or contingent reinforcement.

What is a placebo?

The term placebo is most frequently used to refer to the ingestion of an inert substance. The concept,

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URL: http://www.ursri.com.

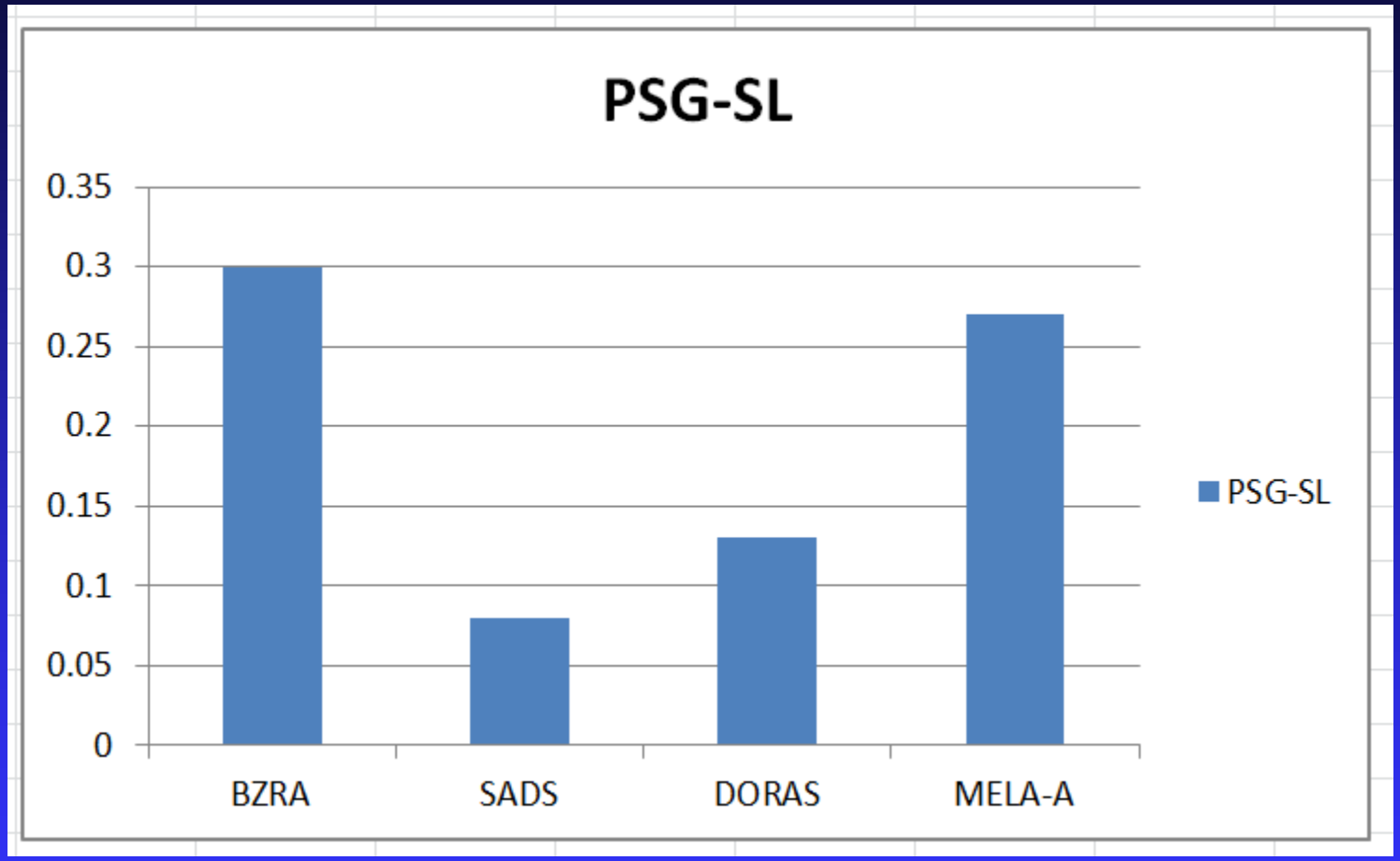
[1] The term sleep continuity is used to represent one of the two major classes of sleep variables (sleep continuity vs sleep architecture measures) and denotes the set of variables that are associated with sleep initiation and maintenance (sleep latency, number of awakenings, wake after sleep onset and total sleep time).

1087-0792/$ - see front matter © 2005 Published by Elsevier Ltd.
doi:10.1016/j.smrv.2005.05.001

RELATIVE EFFICACY:
A META-ANALYTIC WORK IN PROGRESS

A COLLABORATIVE WORK WITH
LIZ CULNAN MS, SUHAIB KHADER BA, CHIARA BAGLIONI PHD AND DIETER RIEMANN PHD

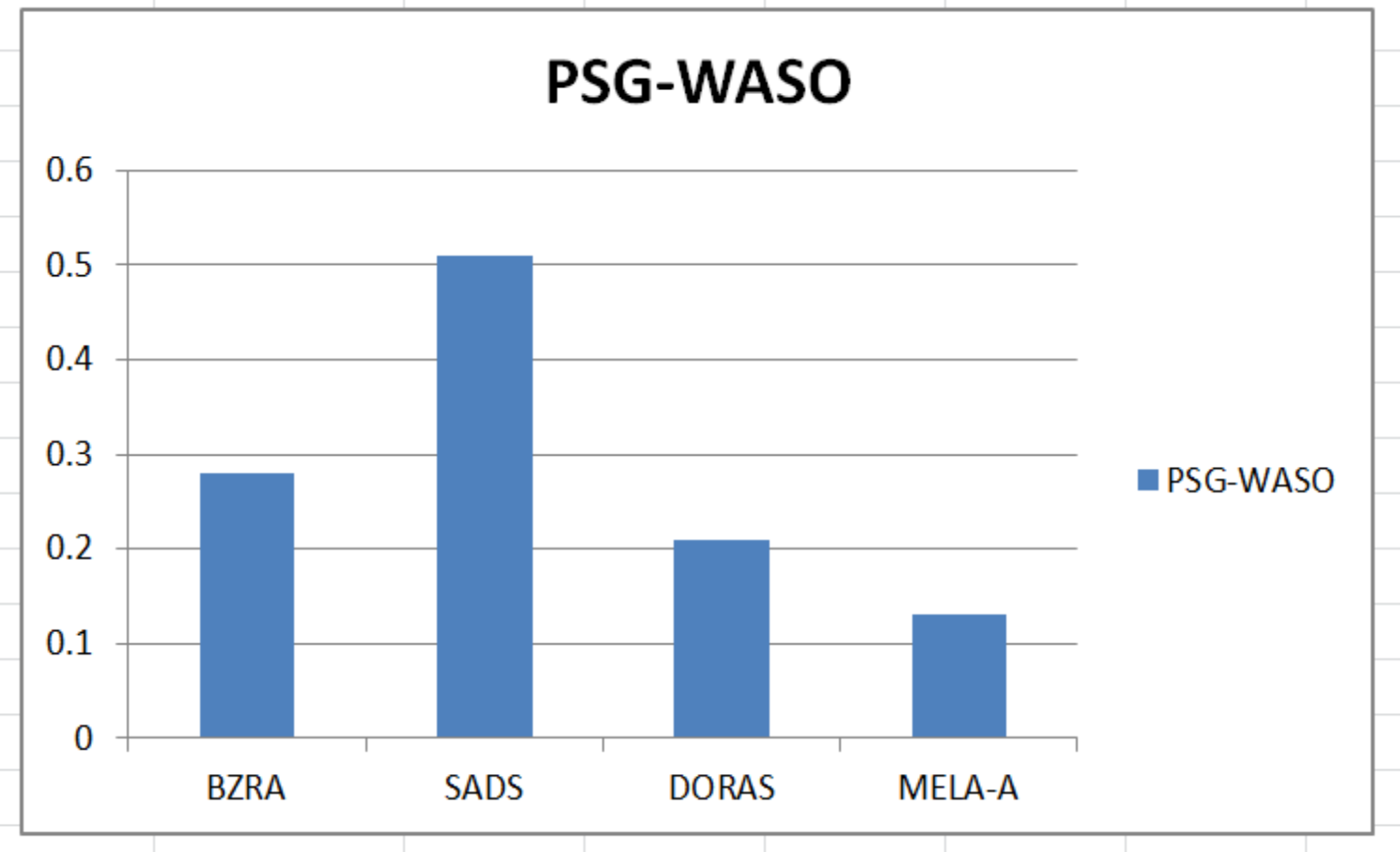
RELATIVE EFFICACY ON PSG



NOTE: ESs PRE-POST CHANGE ADJUSTED FOR PLACEBO EFFECTS

DORAs ARE BASED SOLEY ON
ALMOREXANT DATA (50mg & 100mg)

RELATIVE EFFICACY ON PSG



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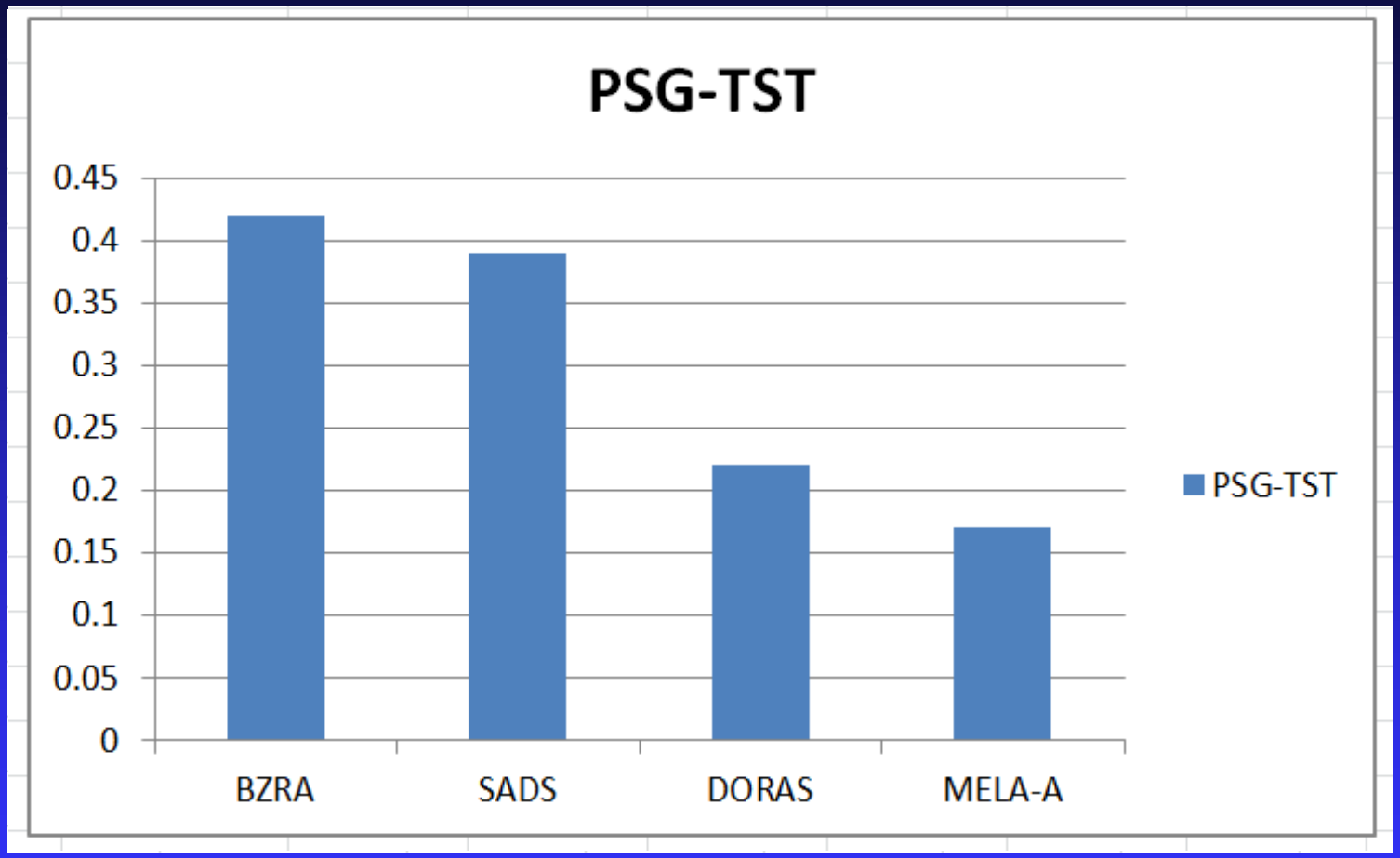

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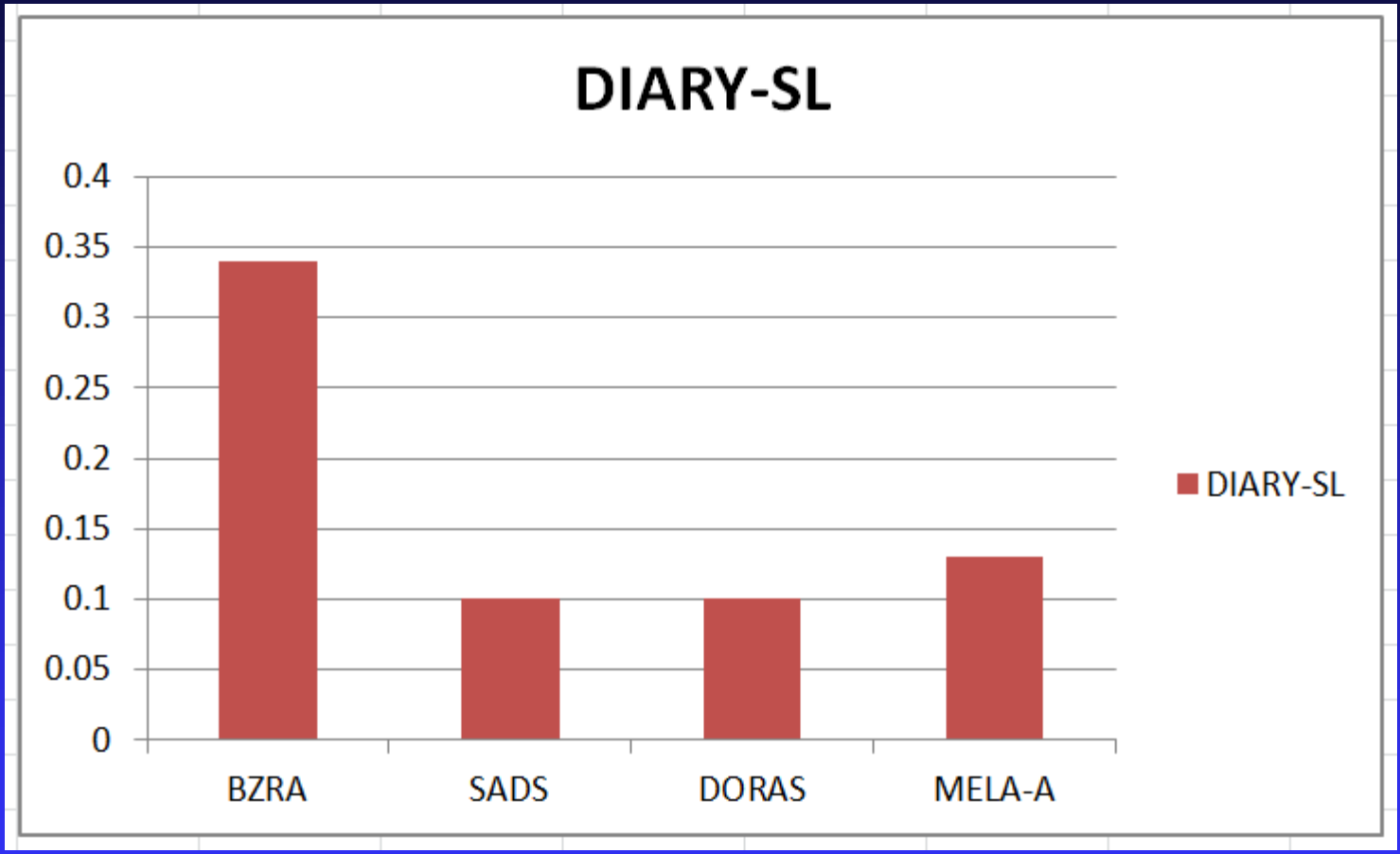
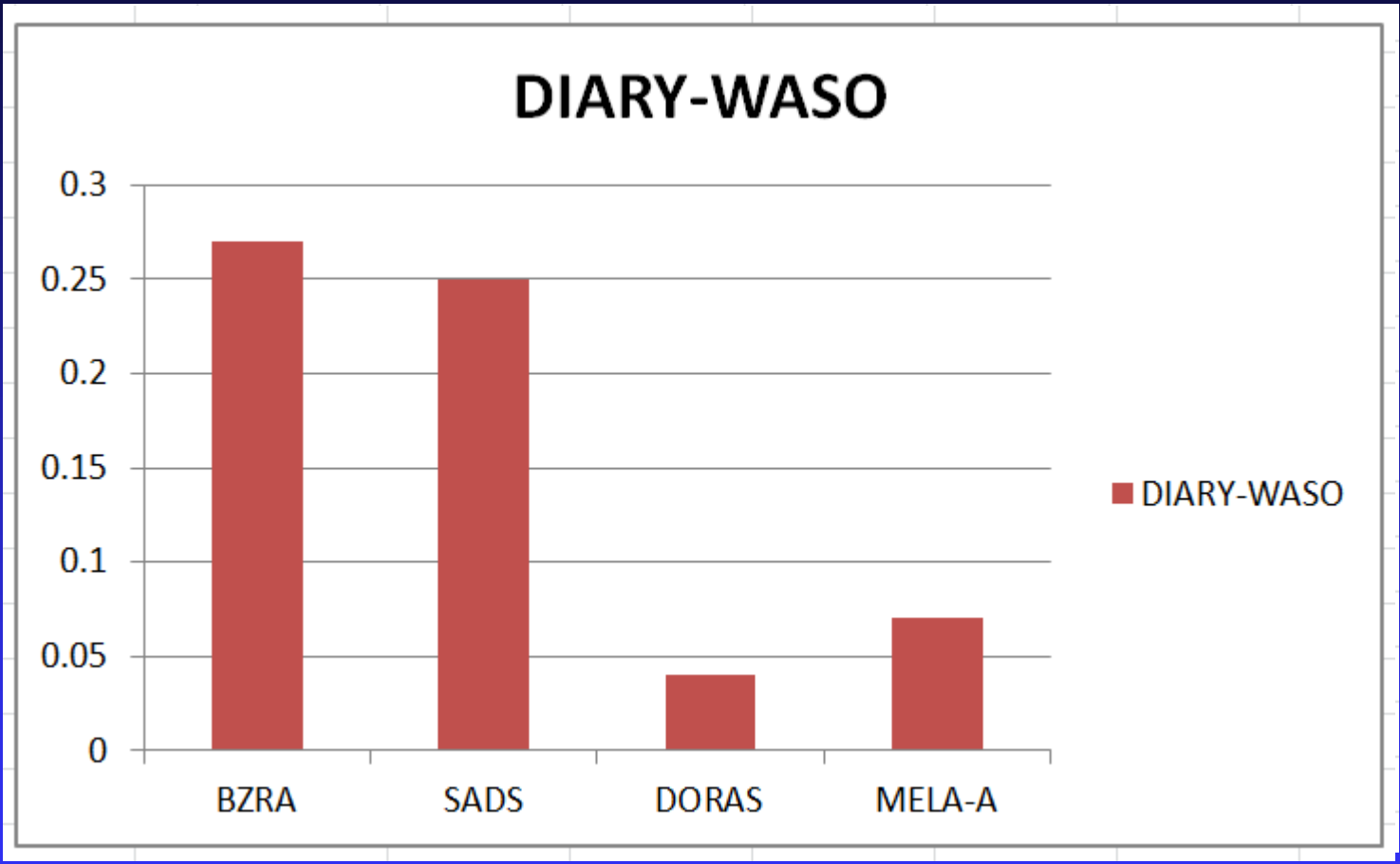
RELATIVE EFFICACY ON DIARIES



RELATIVE EFFICACY ON DIARIES



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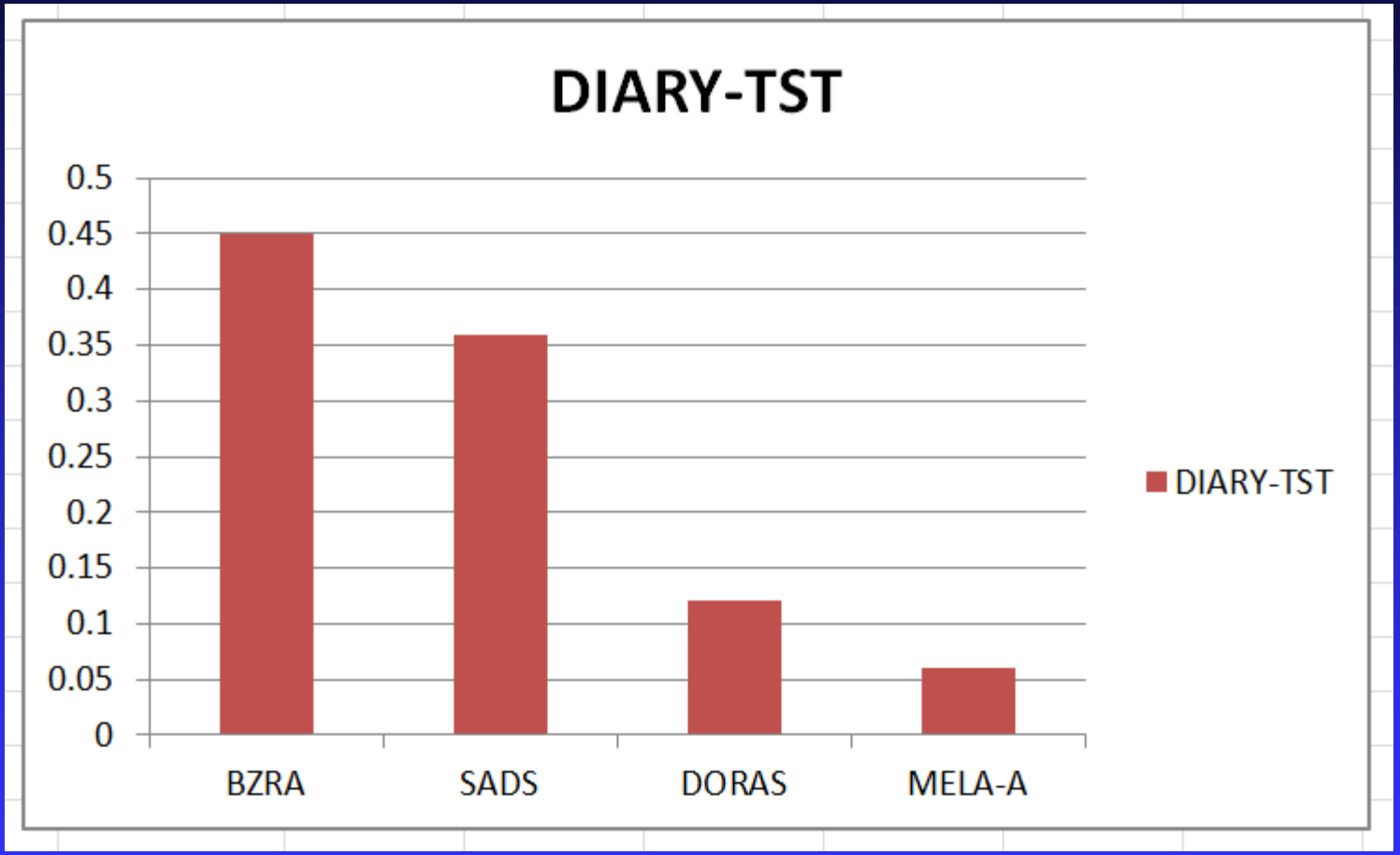
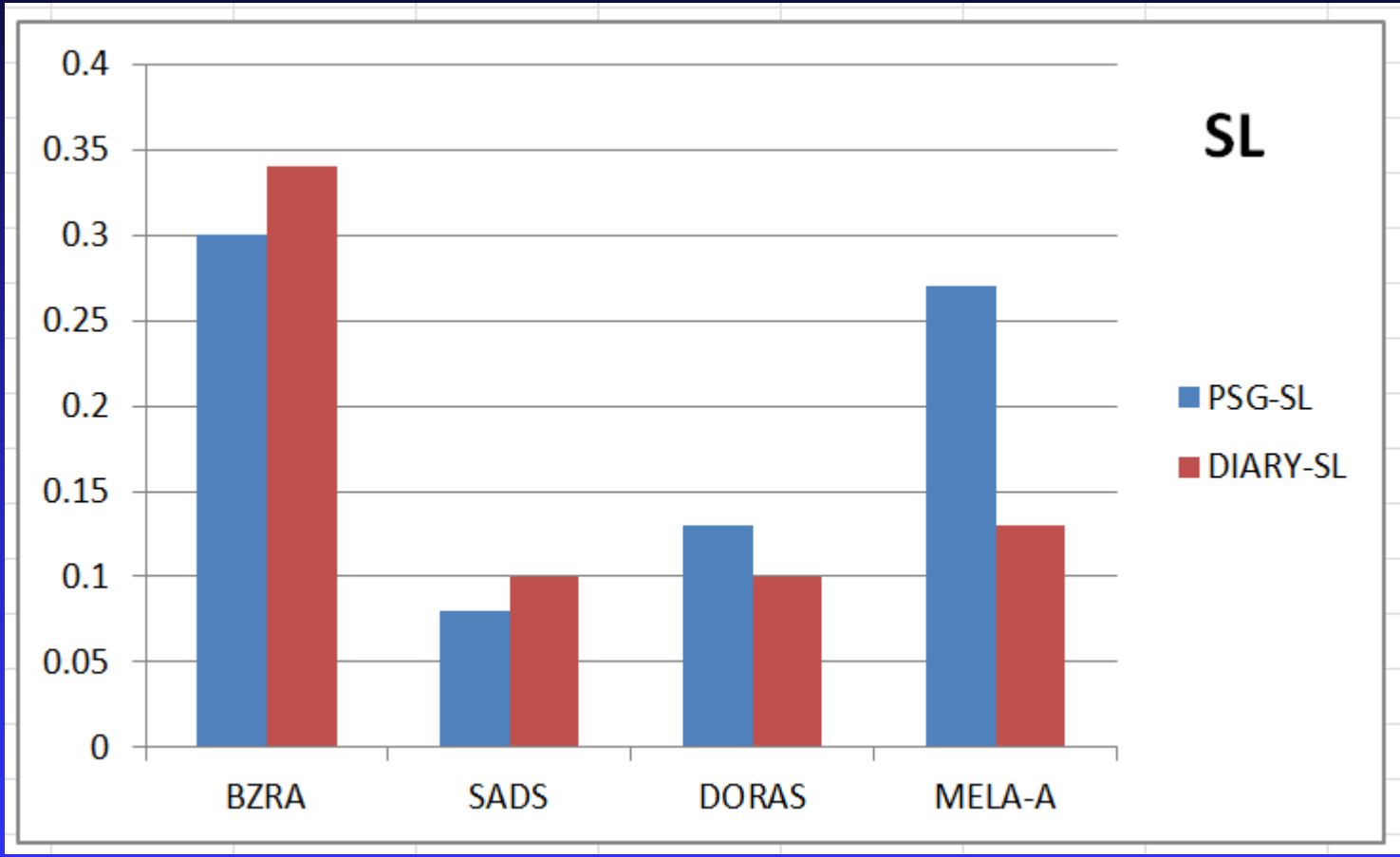


AND NOW THE

RELATIVE EFFICACY



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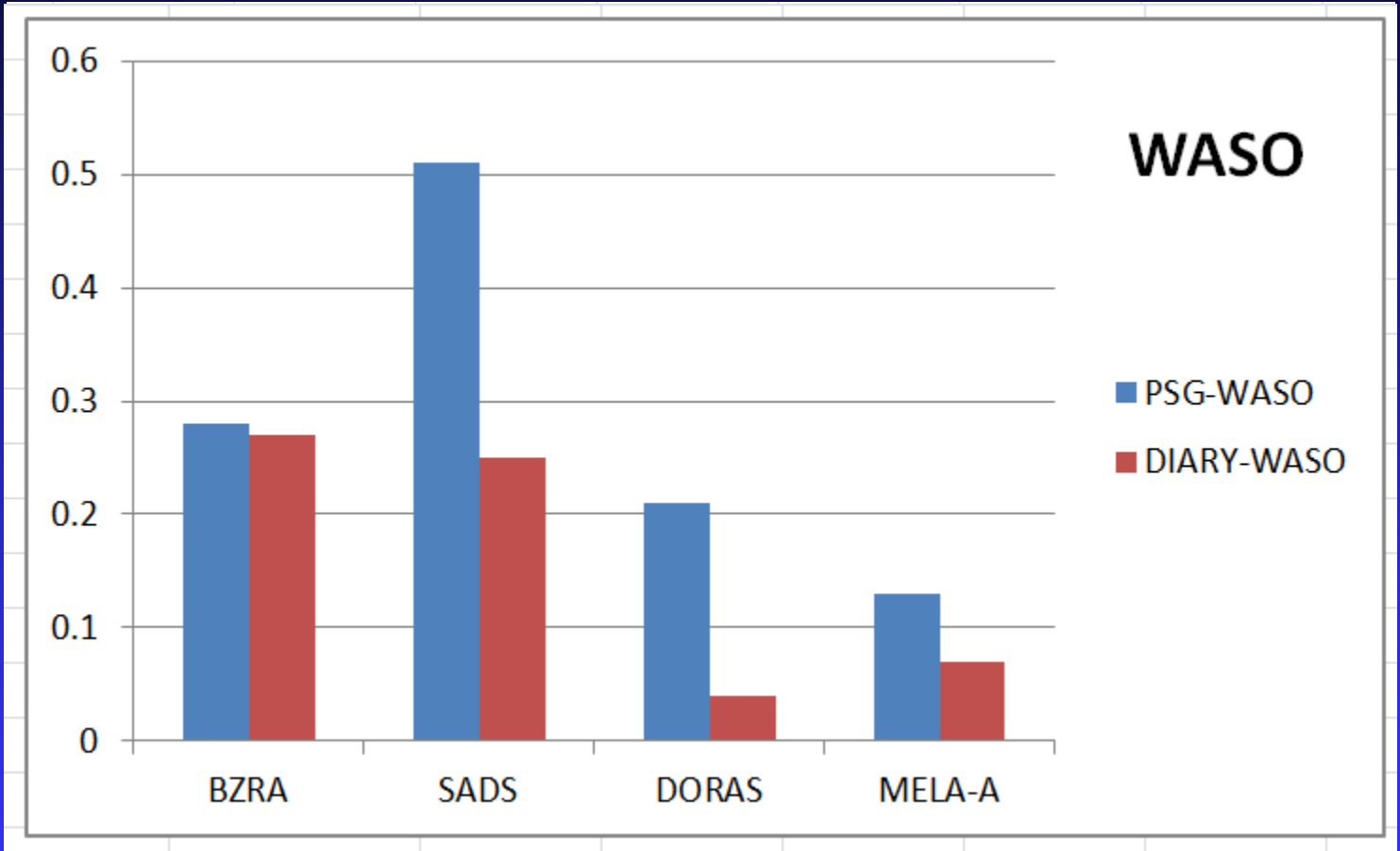

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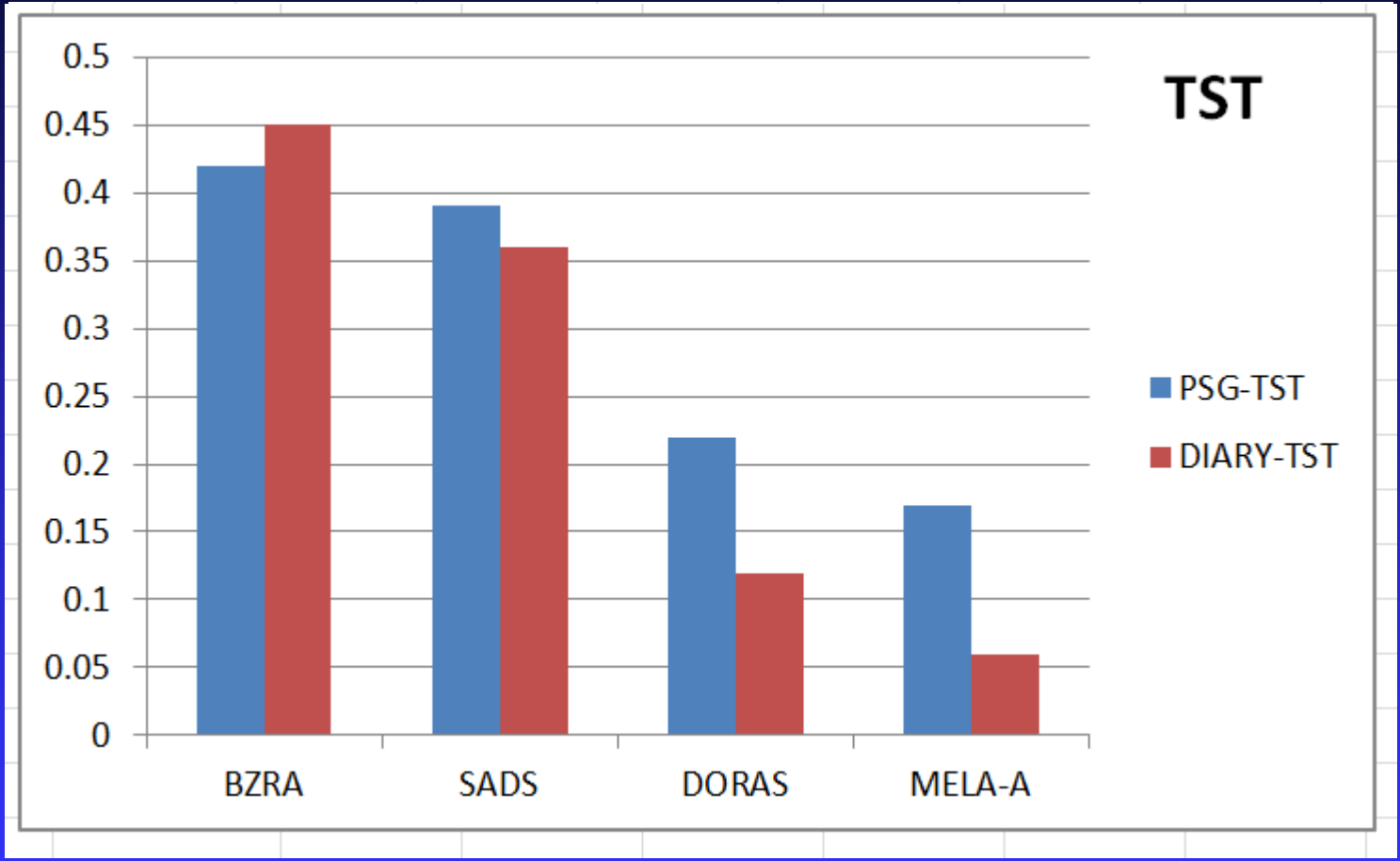

BOTTOM LINE

THE LARGEST MOST PERCEPTIBLE EFFECTS ARE WITH BZRAs

TWO MORE THOUGHTS ABOUT MEDS

MAYBE ITS NOT ALL ABOUT THE MEDICATION
MAYBE ITS ABOUT THE REGIMEN !

CURRENT PERSPECTIVE

INSOMNIA IS LIKE CHRONIC PAIN AND HYPNOTICS ARE LIKE OPIOIDS

WHAT IF

INSOMNIA IS LIKE INFECTION AND HYPNOTICS ARE LIKE ANTIBIOTICS?

WHAT WOULD BE THE THERAPEUTIC IMPLICATIONS OF THIS ALTERNATIVE PERSPECTIVE ?

WHAT ABOUT PATIENT PREFERENCE ?

IS THERE SOMETHING TO BE LEARNED BY SYSTEMATICALLY ASSESSING PREFERNCE?

NOT EVERYONE, HOWEVER, IS KEEN ON HYPNOTICS

The Dark Side of Sleeping Pills

By Daniel F. Kripke, M.D.*

Click to watch Dr. Kripke's September 2008 UCSD presentation, "How Sleeping Pills Can Harm You"

AND NOW A WORD FROM
OUR SPONSOR

When proven ineffective, the sandman is replaced by the boulder guy

PLUS & MINUSES FOR EACH TREATMENT MODALITY

Behavior Therapy

+ Good “short” & long term efficacy
+ No issues re: drug interactions (?)
+ Does not alter sleep architecture (or maybe it does)
+ No rebound insomnia
+ No abuse potential
+ No issues re: LD

- Takes between 3 - 8 weeks (Latency!)
- Transient worsening of symptoms (1-2 weeks)
- Requires substantial patient compliance
- Only effective as practiced by specialists (?)

J. Sleep Res. (2004) 13, 385–393

Effect of cognitive behavioural therapy for insomnia on sleep architecture and sleep EEG power spectra in psychophysiological insomnia

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Accepted in revised form 14 September 2004; received 28 November 2003

SUMMARY There is now an overwhelming preponderance of evidence that cognitive behavioural therapy for insomnia (CBT-I) is effective, as effective as sedative hypnotics during acute treatment (4–8 weeks), and is more effective in long term (following treatment). Although the efficacy of CBT-I in the treatment of chronic insomnia is well known, however there is little objective data on the effects of CBT-I on sleep architecture and sleep EEG power densities. The present study evaluated, first, subjective change in sleep quality and quantity, and secondly the modifications occurring in polysomnography and EEG power densities during sleep after 8 weeks of CBT-I. Nine free drug patients with psychophysiological insomnia, aged 33–62 years (mean age 47 ± 9.7 years), seven female and two male participated in the study. Self-report questionnaires were administered 1 week before and 1 week after CBT-I, a sleep diary was completed each day 1 week before CBT-I, during CBT-I and 1 week after CBT-I. Subjects underwent two consecutive polysomnographic nights before and after CBT-I. Spectral analysis was performed the second night following 36 h of controlled wakefulness. After CBT-I, only scales assessing insomnia were significantly decreased, stages 2, REM sleep and SWS durations were significantly increased. Slow wave activity (SWA) was increased and the SWA decay shortened, beta and sigma activity were reduced. In conclusion CBT-I improves both subjective and objective sleep quality of sleep. CBT-I may enhance sleep pressure and improve homeostatic sleep regulation.

KEYWORDS beta activity, cognitive behavioural therapy, insomnia questionnaires, psychophysiological insomnia, polysomnography, sigma activity, slow wave activity

INTRODUCTION

Insomnia is among the most frequent health complaints among adults (Hajak, 2000; Morin and Kwentus, 1988). Epidemiological surveys indicate that between 9 and 15% of the population complain of chronic insomnia (Bixler *et al.*, 1979; Ford and Kamerow, 1989; Gallup Organization, 1991, 1995; Mellinger *et al.*, 1985). According to DSM IV [American Psychiatric Association (APA), 1994] psychophysiological insomnia (PPI) is a form of primary insomnia and is found in about 12.5–15% of insomniac patients (Buysse *et al.*, 1994; Coleman *et al.*, 1982). PPI is defined by the International Classification of Sleep Disorders (ICSD, 1997) as a chronic (generally at least 6 months) subjective difficulty in initiating or maintaining sleep and a feeling of non-restorative sleep occurring at least three nights a week. PPI starts in adulthood and is not associated with either pathology or substance abuse.

The aetiology of PPI is complex implicating both psychological and physiological explanatory factors. Psychological, cognitive (dysfunctional beliefs and attitudes about sleep) and

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Sleep parameters	*Before CBT-I*	*After CBT-I*	*Z*	*P*	*ES*
Sleep architecture					
Total sleep time (min)	323.67 (37.32)	415.39 (41.35)	−2.67	0.008	−2.22
Stage 1 (min)	37.50 (15.96)	39.50 (12.68)	−0.77	0.441	−0.13
Stage 1 (%)	11.67 (4.92)	9.59 (3.15)	−1.95	0.051	0.48
Stage 2 (min)	168.39 (33.35)	206.06 (46.60)	−2.67	0.008	−0.89
Stage 2 (%)	52.37 (11.26)	49.36 (8.99)	−2.67	0.008	0.28
Slow wave sleep (min)	62.56 (33.30)	86.50 (23.77)	−2.55	0.01	−0.79
Slow wave sleep (%)	19.09 (9.21)	21.21 (7.13)	−1.24	0.214	−0.24
REM sleep (min)	55.22 (20.33)	83.33 (23.63)	−2.24	0.025*	−1.21
REM sleep (%)	16.87 (5.11)	19.85 (4.97)	−1.60	0.110	−0.56

Values in parenthesis represent SD. ES, effect size.

DOES THIS STUFF WORK ?

There is now an overwhelming preponderance of evidence that Cognitive Behavioral Therapy for insomnia (CBT-I) is efficacious, effective, as efficacious as sedative hypnotics during acute treatment (4-8 weeks), and is more efficacious in the long term (following treatment)

Put differently, CBT-I produces a treatment response in 60% of patients and nearly 65% of treatment responders attain remission after treatment has been discontinued

EFFICACY – 9 META-ANALYSES

Author	Year	Title	Journal
Morin et al.	1994	Nonpharmacological interventions for insom-nia: a meta-analysis of treatment efficacy	Am J Psychiatry, 151, 1172-1180
Murtagh & Greenwood	1995	Identifying effective psychological treatments for insomnia: a meta-analysis	J Consult Clin Psychol, 1995, 79-89
Pallesen et al.	1998	Nonpharmacological interventions for insom-nia in older adults: a meta-analysis of treat-ment efficacy	Psychotherapy, 35, 472-481
Montgomery & Dennis	2003	Cognitive behavioral interventions for sleep problems in adults aged 60+	Cochrane Library, 1, 1-39/ Sleep Med Rev,8, 47-62
Irwin et al.	2006	Comparative meta-analysis of behaviroral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+ years of age	Health Psychology, 25,3-14.
Okajima et al.	2011	A meta-analysis on the treatment effective-ness of cognitive behavioral therapy for primary insomnia	Sleep & Biol Rhythms, 9,24-34
Mitchell et al	2012	Comparative effectiveness of cognitive behavioral therapy for insomnia: a systematic review	BMC Family Practice, 13, 40-51
Miller et al.	2014	The evidence base of sleep restriction therapy for treating insomnia disorder	Sleep Med Rev, 18, 415-424
Koffel et al.	2015	A meta-analysis of group cognitive behaviorla therapy for insomnia	Sleep Med Rev, 19 epub

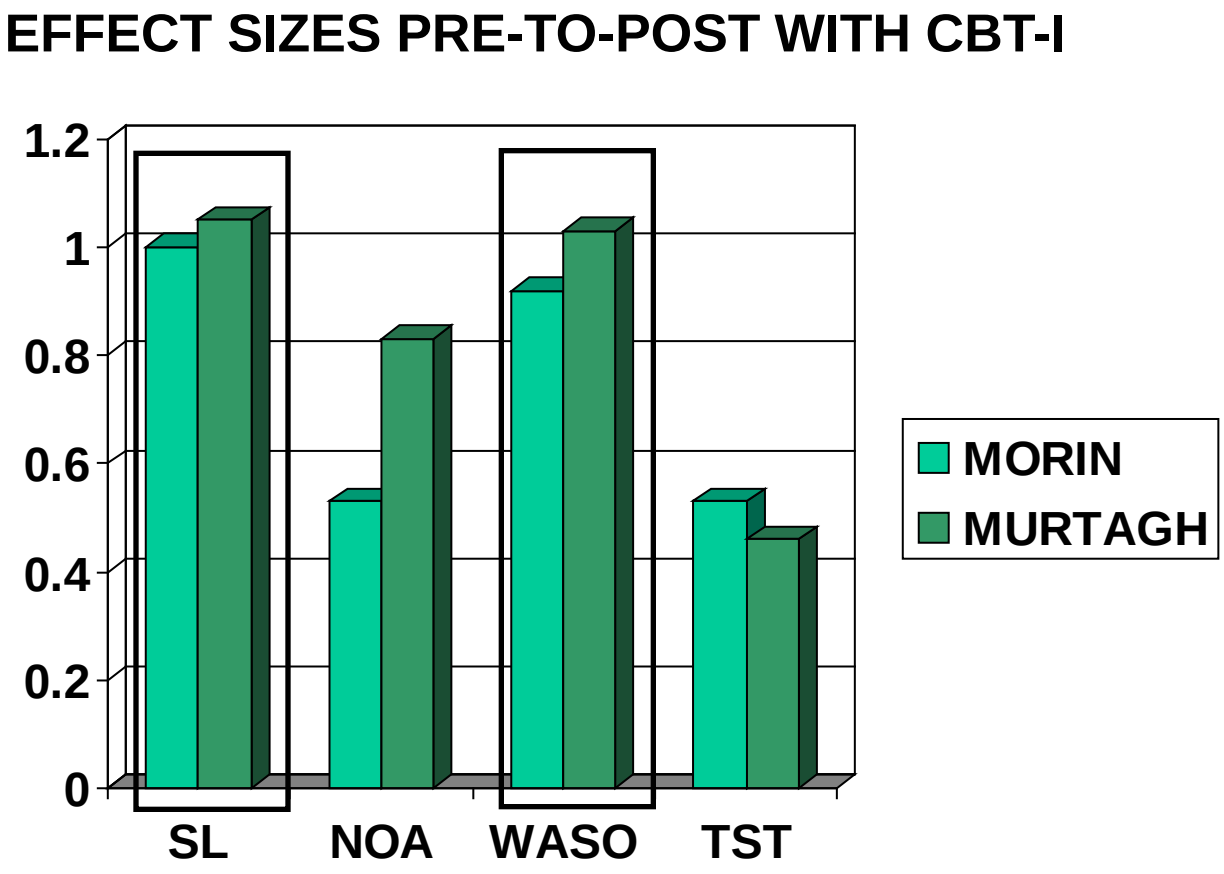
Compiled by Dieter Riemann

EFFICACY

Morin et al.
Nonpharmacological interventions for insomnia: a meta-analysis of treatment efficacy.
Am J Psychiatry 1994; 151(8):1172-1180.

Murtagh et al.
Identifying effective psychological treatments for insomnia: a meta-analysis.
J Consult Clin Psychol 1995; 63(1):79-89.

EFFICACY


EFFECT SIZES PRE-TO-POST WITH CBT-I
1.2
1
0.8
0.6
0.4
0.2
0
SL
NOA
WASO
TST
MORIN
MURTAGH

RCT DATA AIN'T THE REAL WORLD !

RCT

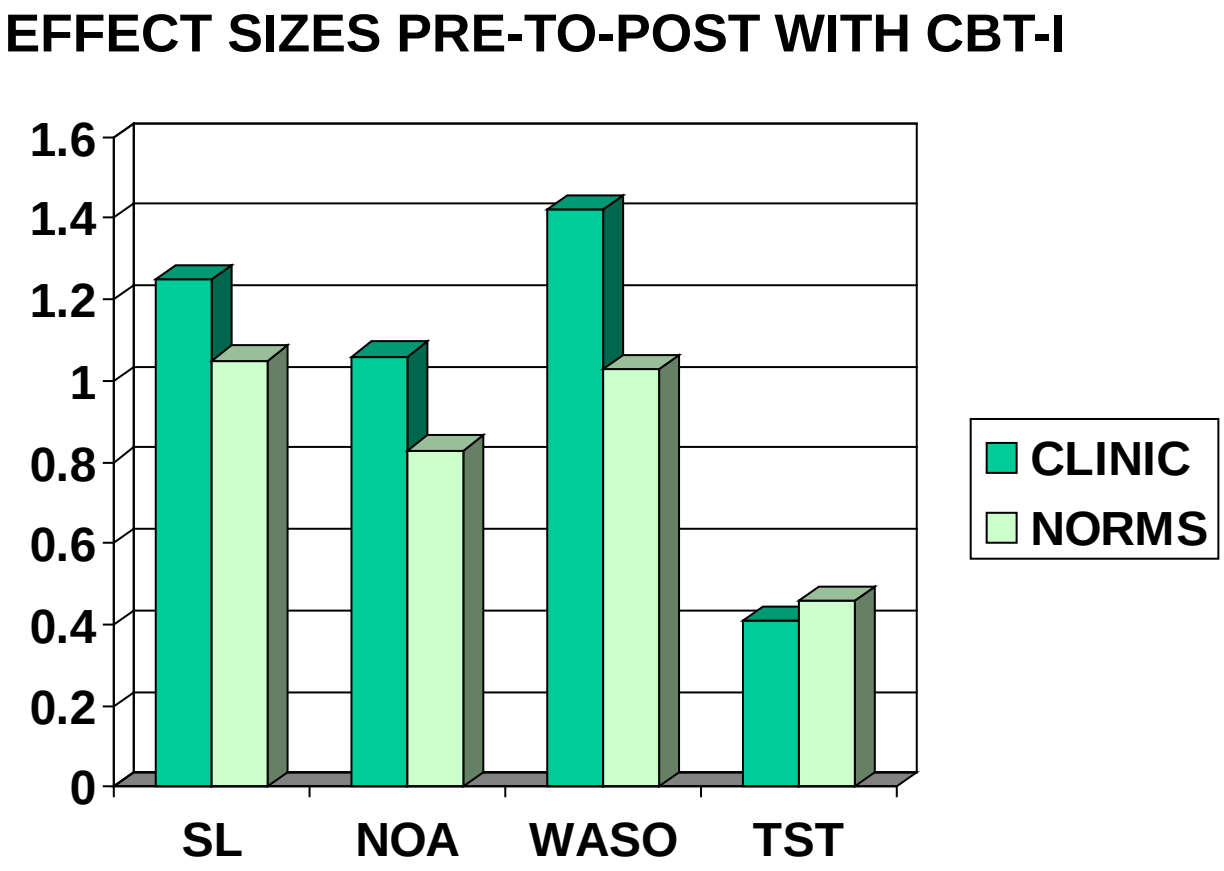
CLINIC

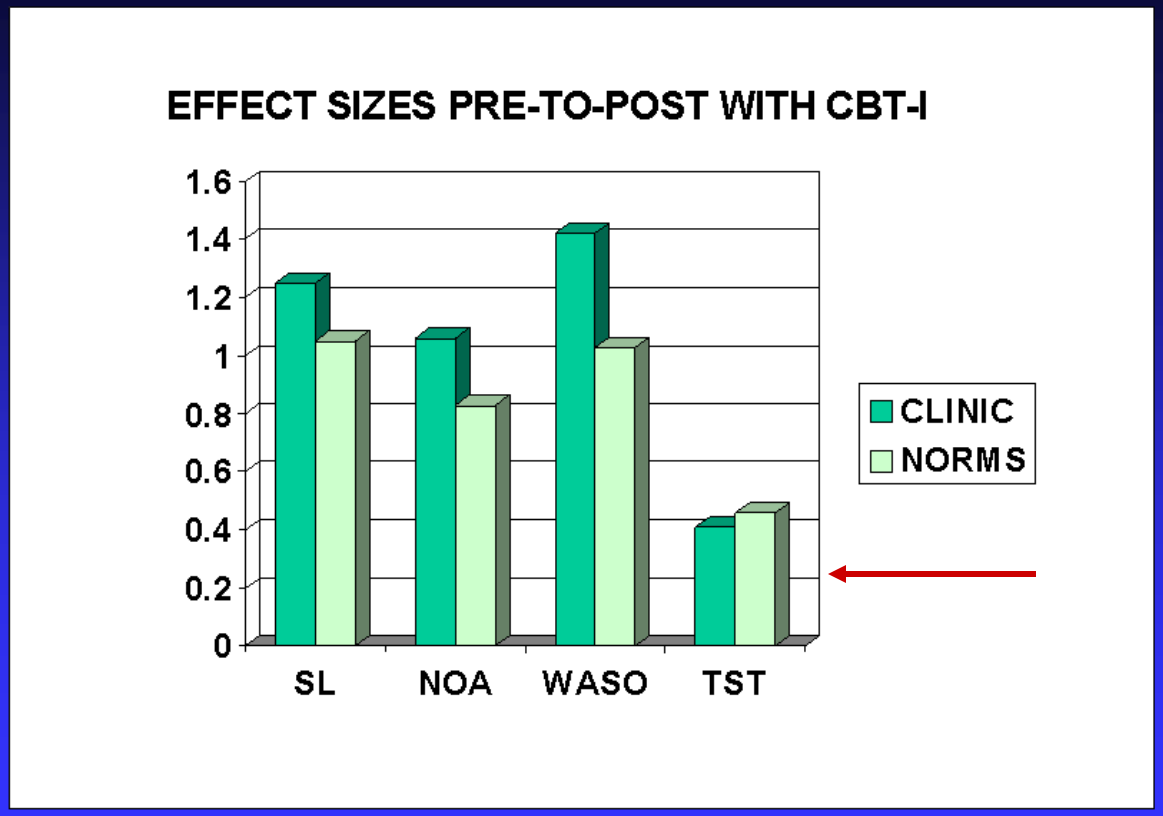
EFFECTIVENESS

AN EXAMPLE

Perlis, M, Aloia M, Boehmler J, Millikan A, Greenblatt D, Giles D. Behavior treatment of insomnia: a clinical case series study. The Journal of Behavioral Medicine,23(2)149-161, 2000.

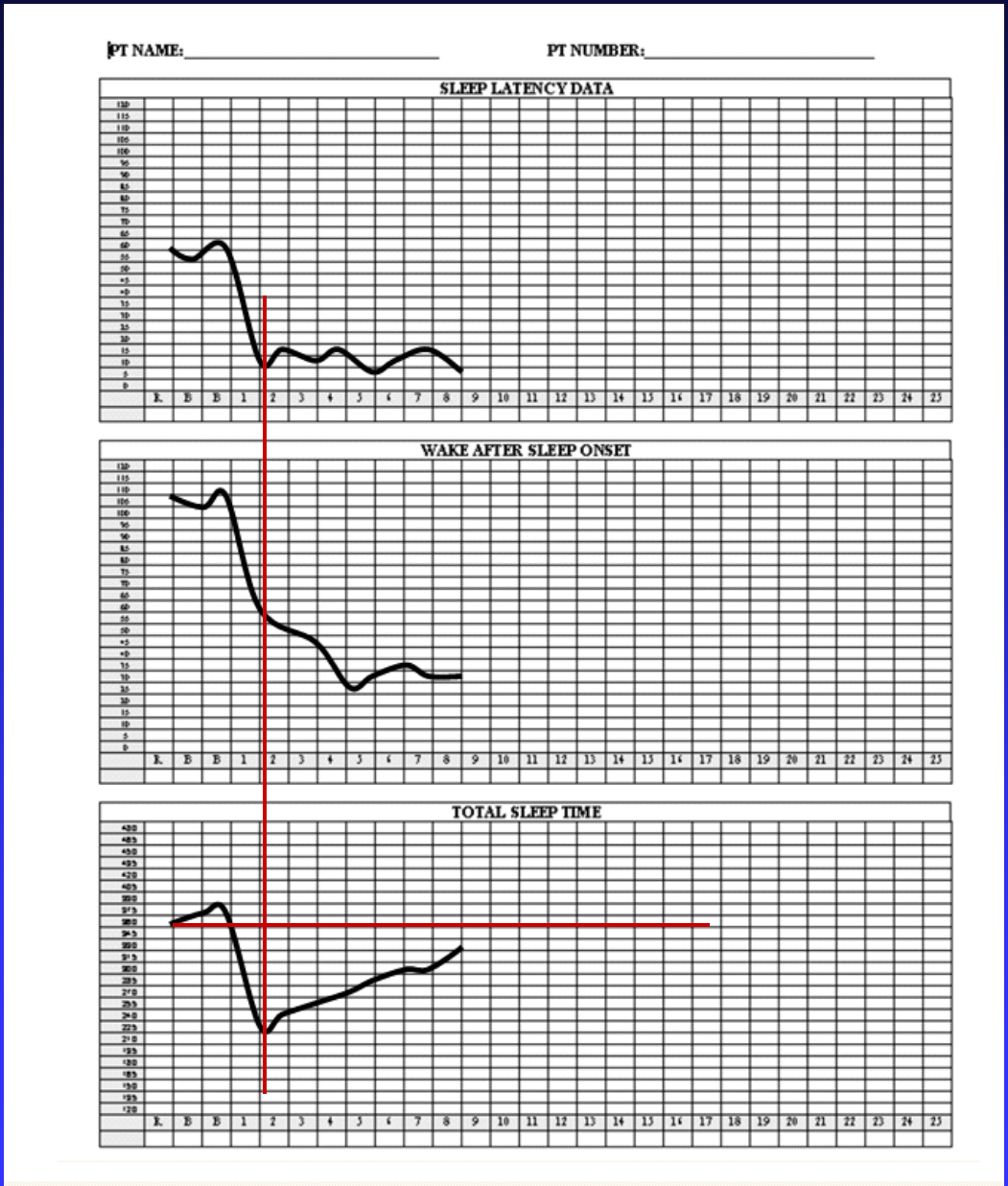
EFFECTIVENESS





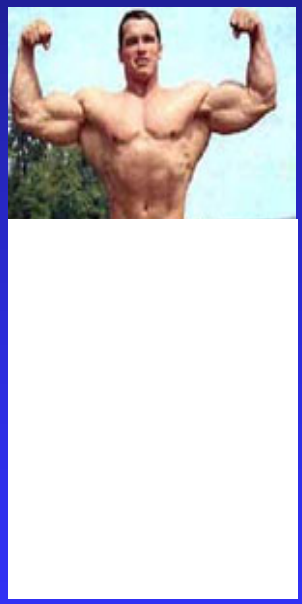
WHY ARE THE TST EFFECTS SO POOR ?

HERE'S WHY

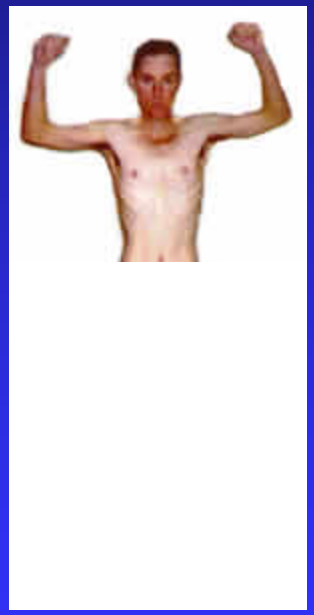

PT NAME:
PT NUMBER:
SLEEP LATENCY DATA
WAKE AFTER SLEEP ONSET
TOTAL SLEEP TIME

HOW DOES PHARMACOTHERAPY WITH BZRAs COMPARE WITH COGNITIVE BEHAVIORAL THERAPY?

IS THIS AN ACCURATE REPRESENTATION ?

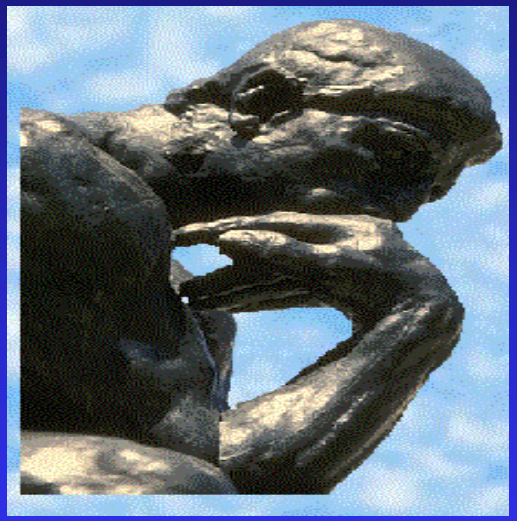


MEDICAL TX FOR INSOMNIA



CBT TX FOR INSOMNIA

I THINK NOT



RELATIVE EFFICACY

HOW DO MEDICAL AND BEHAVIORAL INTERVENTIONS COMPARE ?

Studies comparing CBT-I to pharmacological therapies

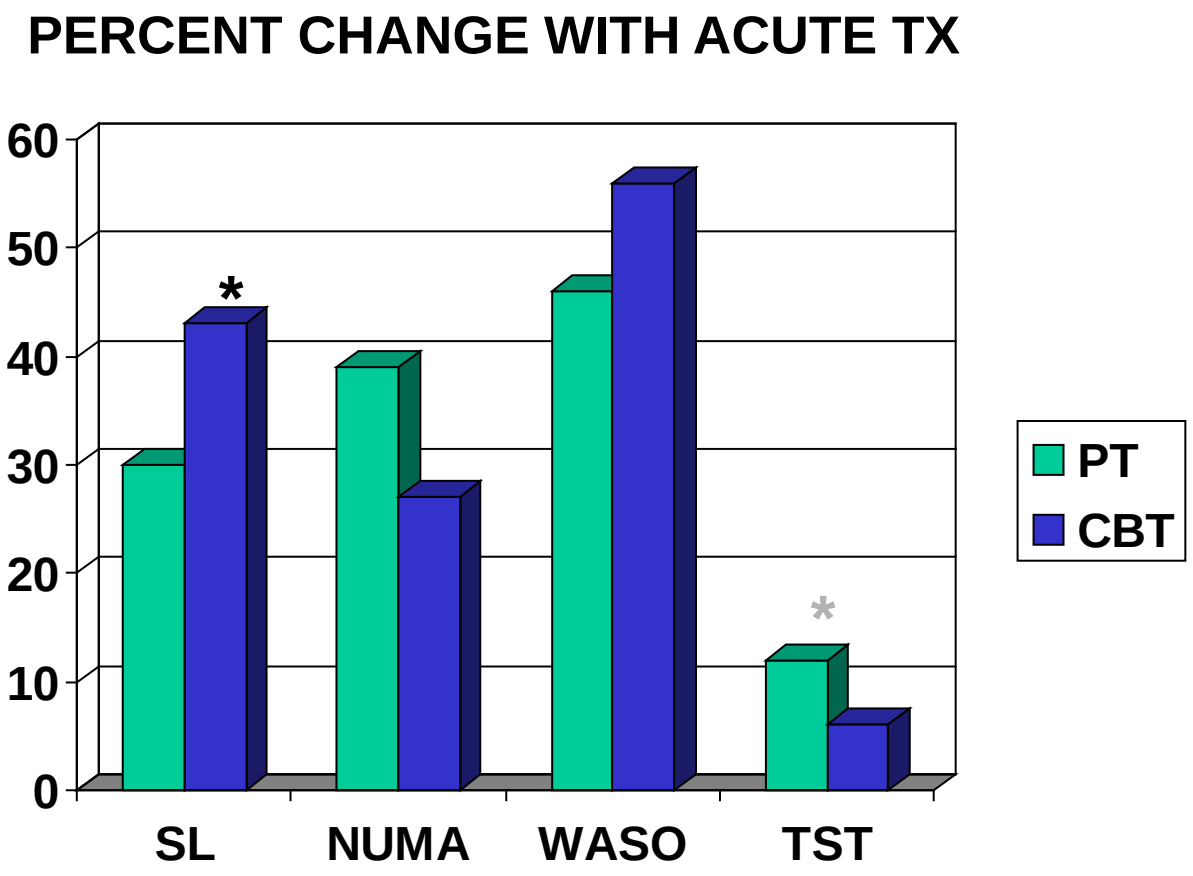
Study Location	Design Quality	Patients Longest follow-up	Intervention and duration	Comparison	Sleep measurements reported	Comment
CBT-I vs. zopiclone						
Sievertsen 2006 (26) Norway	RCT 5	46 patients, age 55 and up 12 months	Individual CBT-I, 6 weekly sessions	Zopiclone, 7.5 mg nightly	Sleep diaries, polysomnography	Study also included placebo group Daytime outcomes reported in (36)
CBT-I vs. zolpidem						
Jacobs 2004 (27) USA	RCT 5	63 patients, age 25-64 12 months	Individual CBT-I, 5 sessions, 6 weeks; plus 1 telephone session	Zolpidem, see comment	Sleep diaries, sleep monitor	Dose 10 mg→5 mg→5 mg q2d
CBT-I vs. temazepam						
Wu 2006 (29) China	RCT 2	77 patients 8 months	Individual CBT-I 2 per week, 8 weeks	Temazepam, see comment	Sleep diaries, polysomnography	Dose 7.5 mg→30 mg→15 mg Study also included placebo and combined therapy groups
Morin 1999 (28) Canada	RCT 6	78 patients, age 55 and up 24 months	Group CBT-I 8 weekly sessions	Temazepam, see comment	Sleep diaries, polysomnography	Dose 7.5 mg→30 mgas needed Study also included placebo and combined therapy groups. Adverse effects reported in (37) Attitudes reported in (38)
CBT-I vs. triazolam						
McCluskey 1991 (30) USA	RCT 4	30 patients 9 weeks	Group CBT-I 2 per week, 3 weeks	triazolam, 0.5 mg then tapered to 0	Sleep diaries	triazolam group also had weekly group meetings but no CBT-I

RELATIVE EFFICACY

HOW DO MEDICAL AND BEHAVIORAL INTERVENTIONS COMPARE ?

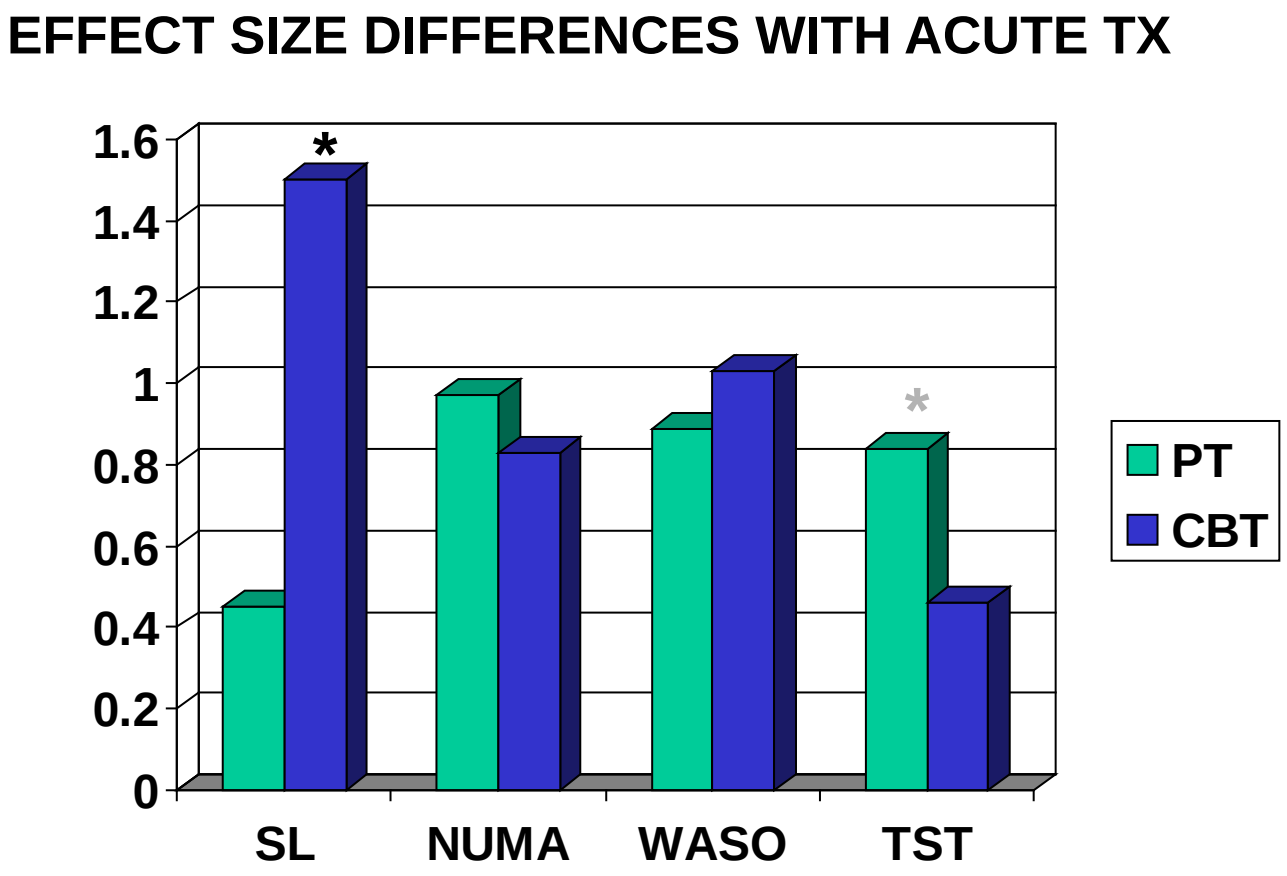
Smith MT, Perlis, ML, Park A, Giles DE, Pennington JA, Buysse, D. Behavioral treatment vs pharmacotherapy for Insomnia - A comparative meta-analyses. American Journal of Psychiatry. 159: 5-11. 2002.

COMPARATIVE EFFICACY



SMITH, PERLIS, ET AL., 2002

COMPARATIVE EFFICACY



CBT & PCT HAVE "EQUIPOTENCY" IN SHORT RUN

AND

CBT HAS BETTER EFFICACY IN THE LONG RUN

(MAYBE – ASK AT BREAK)

WHAT ABOUT MODE OF DELIVERY ?

Journal of Consulting and Clinical Psychology
2004, Vol. 72, No. 4, 653–659

0022-006X/04/$12.00 DOI: 10.1037/0022-006X.72.4.653

Cognitive–Behavioral Therapy for Insomnia: Comparison of Individual Therapy, Group Therapy, and Telephone Consultations

Célyne H. Bastien, Charles M. Morin, Marie-Christine Ouellet, France C. Blais, and Sébastien Bouchard
Université Laval, Québec

Forty-five adults with primary insomnia received cognitive–behavioral therapy (CBT) implemented in a group therapy format, in individual face-to-face therapy or through brief individual telephone consultations. The results indicate that CBT was effective in improving sleep parameters with all 3 methods of treatment implementation, and there was no significant difference across methods of implementation. All 3 treatment modalities produced improvements in sleep that were maintained for 6 months after treatment completion. These results suggest that group therapy and telephone consultations represent cost-effective alternatives to individual therapy for the management of insomnia.

About 30% of adults will suffer from sleep difficulties during the course of a year, including about one third (10% of the population) who will report chronic difficulties in falling or staying asleep (Ford & Kamerow, 1989). Insomnia is more prevalent among women, older adults, and among persons suffering from medical (Blais, Morin, Boisclair, Granger, & Guay, 2001; Hohagen et al., 1993) or psychological conditions (Buysse et al., 1994). It is often accompanied by daytime complaints (attention–concentration problems, mood disturbances, or fatigue), which can significantly diminish one's quality of life (Chilcott & Shapiro, 1996). Hypnotics remain the most frequent treatment, despite risks of tolerance and dependence with long-term use (Hauri, 1997).

Psychological interventions have been shown to be as effective as pharmacotherapy in the short-term and more effective in the long-term (McClusky, Milby, Switzer, Williams, & Wooten, 1991; Morin, Colecchi, Stone, Sood, & Brink, 1999). Two meta-analyses have indicated that behavioral interventions produce significant improvements in 70% to 80% of patients with insomnia and that treatment gains are well maintained over time (Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995).

Despite these advantages, nonpharmacological interventions for insomnia remain underused, their use being compromised by their limited accessibility. Money, time, and effort constraints involved in psychotherapy may seem overwhelming in contrast with the apparent simplicity and accessibility of pharmacotherapy. As such, several studies have investigated cost-reducing strategies by using brief consultations, group treatments, or self-help approaches (Alperson & Biglan, 1979; Mimeault & Morin, 1999; Morawetz, 1989; Riedel, Lichstein, & Dwyer, 1995).

Group therapy represents a lower cost alternative to individual therapy. However, as in individual therapy, geographical constraints and costs for transportation remain a significant barrier to using group therapy for insomnia. A slight advantage of the individual over the group modality has been proposed (Lacks, 1991; Morin et al., 1994), but these results are insufficient to declare the superiority of individual therapy.

Self-help therapy is the least expensive and involves no transportation. Mimeault and Morin (1999) reported that a cognitive–behavioral bibliotherapy, with or without professional guidance (telephone support), was efficacious in the short term for the treatment of chronic insomnia. However, adding professional guidance to bibliotherapy produced further improvements. Thus, the presence of a therapist seems to optimize treatment response.

Telephone consultations are another cost-reducing strategy. Recently, a "sleep service line," which offers clinical advice about sleep hygiene and behavioral practices for insomnia, improved sleep in more than 25% of callers (Verbeek, Declerck, Kmuistingh Neven, & Coenen, 2002). Although these recent findings suggest the feasibility of implementing insomnia treatment at a minimal cost, a direct comparison of different treatment implementation methods has not yet been carried out in insomnia research.

The objective of the present study was to determine whether cognitive–behavioral therapy (CBT) for insomnia produces different outcomes when delivered either in individual face-to-face therapy or in less costly modalities such as group therapy or brief individual therapy sessions over the phone.

Method

Participants

The participants were French-speaking individuals recruited via advertisements in local newspapers. The inclusion criteria were (a) to be 18 years of age or older, (b) sleep-onset insomnia or sleep-maintenance insomnia (defined as sleep-onset latency or time awake after sleep onset greater than 30 min per night for a minimum of three nights per week as measured on a sleep diary), (c) a duration of insomnia of at least 6 months, and (d) a complaint of at least one negative daytime effect (e.g., fatigue, impaired functioning, or mood disturbances) attributed to poor sleep. The exclusion criteria were (a) another sleep disorder as evaluated by key questions from

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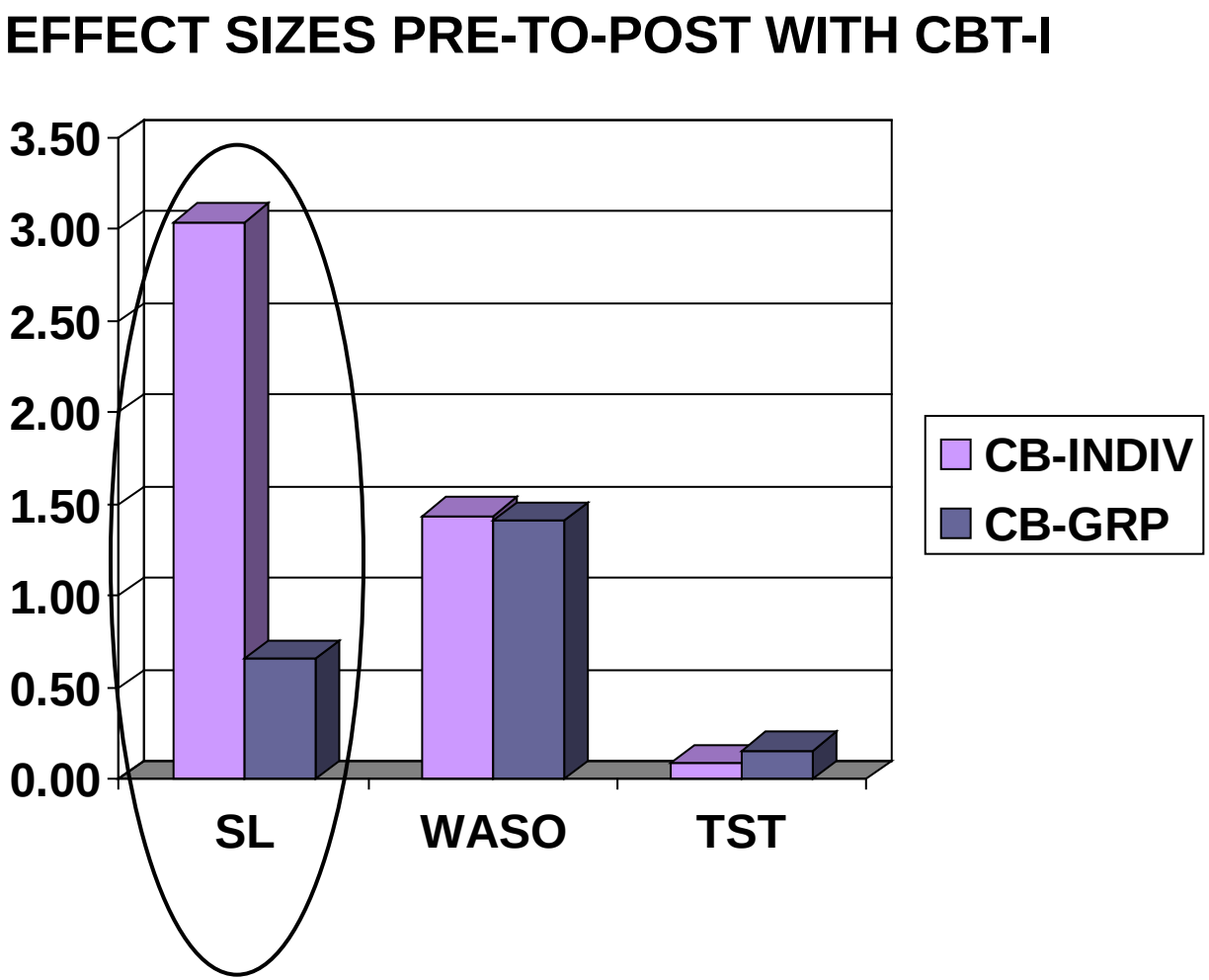
Preparation of this article was supported in part by the National Institute of Mental Health Grant MH55469 and Fonds de la recherché en santé du Québec FRSQ 50433.

Correspondence concerning this article should be addressed to Célyne H. Bastien, École de Psychologie, Université Laval, Ste-Foy, Québec G1K 7P4, Canada. E-mail: celyne.bastien@psy.ulaval.ca

EFFECTIVENESS



Célyne Bastien – JCCP 2004, Vol. 72, No. 4, 653–659


YOU DON'T SAY?

SLEEP AND BIOLOGICAL RHYTHMS

Sleep and Biological Rhythms 2013; **11**: 176–184 doi:10.1111/sbr.12019

ORIGINAL ARTICLE

Comparisons of short-term efficacy between individual and group cognitive behavioral therapy for primary insomnia

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Abstract

The purpose of this study was to compare the efficacy of individual and group cognitive behavioral therapy for insomnia (CBT-I) in outpatients with primary insomnia diagnosed by DSM-IV-TR. The participants were 20 individually treated (I-CBT-I) and 25 treated in a group therapy format (three to five patients per group) (G-CBT-I), which showed no significant difference regarding demographic variables between groups. The same components of CBT-I stimulus control therapy, sleep restriction therapy, cognitive therapy, and sleep hygiene education were applied on both groups. The short-term outcome (4 weeks after treatment) was measured by sleep logs, actigraphy, the Pittsburgh Sleep Quality Index (PSQI), and the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS), and was compared between I-CBT-I and G-CBT-I. The results indicated that CBT-I was effective in improving subjective and objective sleep parameters and subjective sleep evaluations for both individual and group treatment. However, I-CBT-I resulted in significantly better improvements over G-CBT-I, in (i) objective and subjective sleep onset latency time, (ii) objective sleep efficacy and moving time during sleeping, (iii) overall sleep quality and duration of actual sleep time in PSQI, (iv) consequences of insomnia, control and predictability of sleep, sleep requirement expectation, and sleep-promoting practices in DBAS. The present study suggested the superiority of I-CBT-I over G-CBT-I in clinical settings, and further evaluations are necessary.

Key words: behavior and cognition, cognitive behavioral therapy for insomnia, insomnia, primary insomnia, psychology.

INTRODUCTION

Primary insomnia, as defined in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. text version (DSM-IV-TR),[1] is the most common type of chronic insomnia and is almost the same concept as psychophysiological insomnia as defined in the *International Classification of Sleep Disorders* 2nd ed. (ICSD-2).[2]

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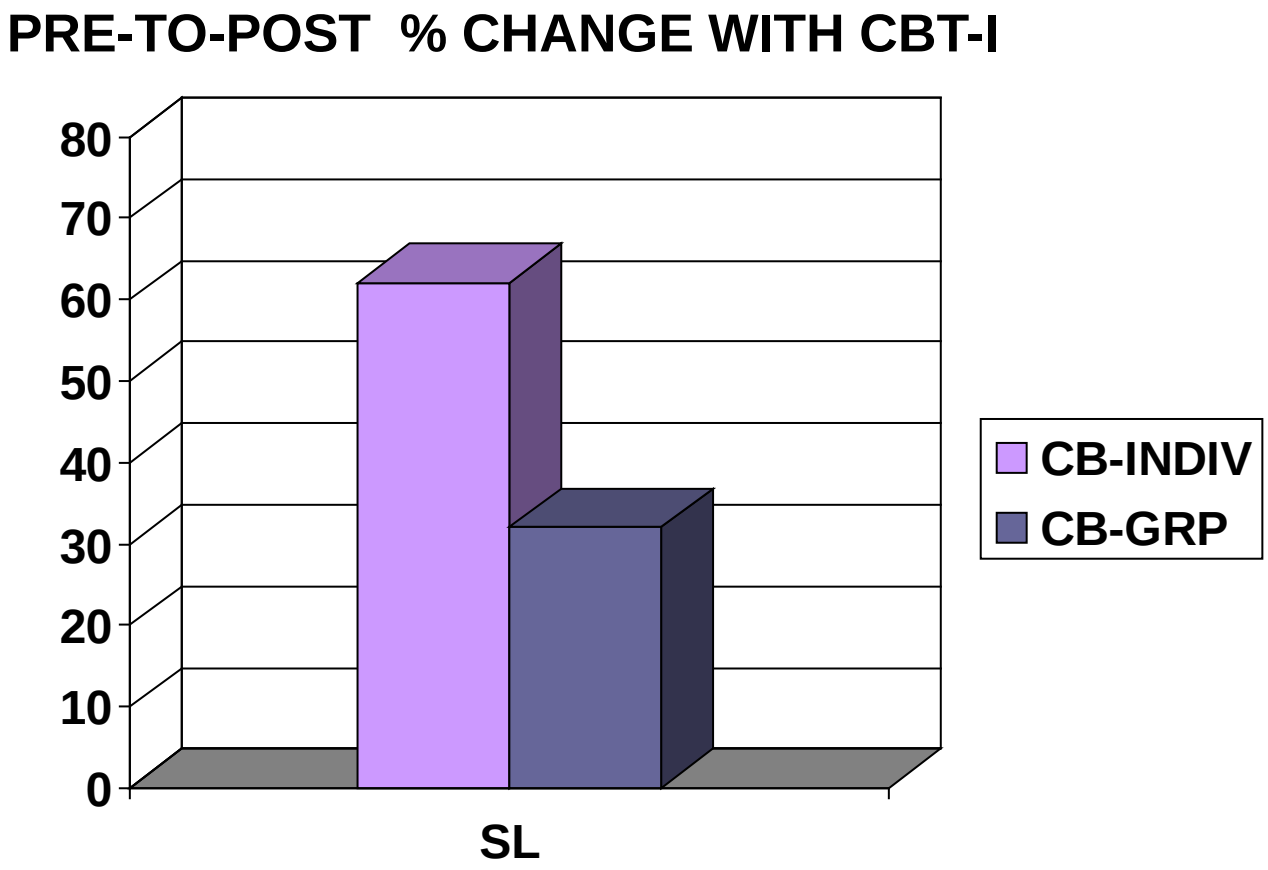
Accepted 29 March 2013.

Primary insomnia is characterized by morbid fear of insomnia, mental arousal, and heightened somatic tension in bed. Recently, it is been emphasized that cognitive behavioral therapy for insomnia (CBT-I) is effective for primary insomnia patients.[3–6]

The best tested and most commonly used method of delivering CBT-I had been via individualized treatment consisting of one-to-one sessions between a therapist and a single patient (I-CBT-I). As providing the I-CBT-I format is a time-consuming and cost-inefficient form of treatment delivery, the most common alternative delivery format is group therapy (G-CBT-I). However, no one established method of G-CBT-I has been used universally.[5,6] Furthermore, whether I-CBT-I and G-CBT-I are

EFFICACY



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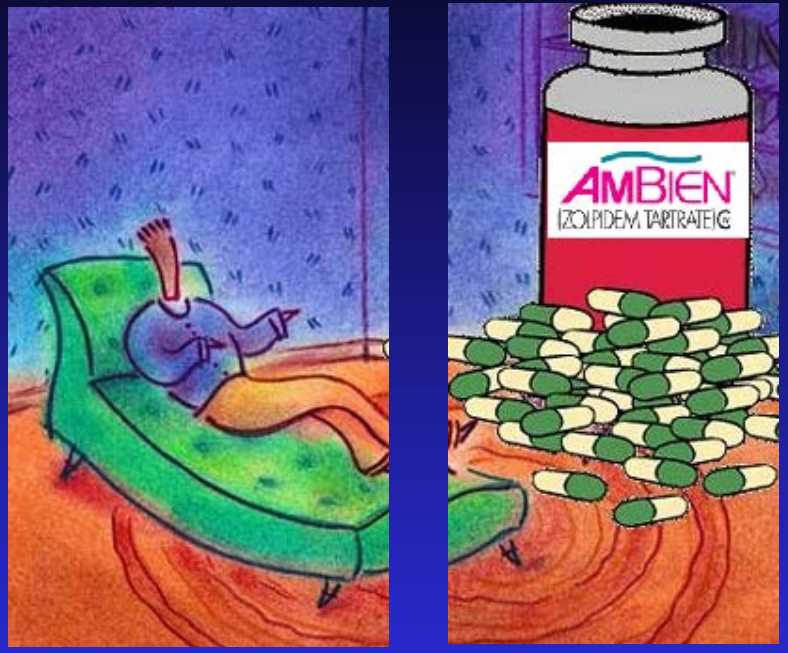
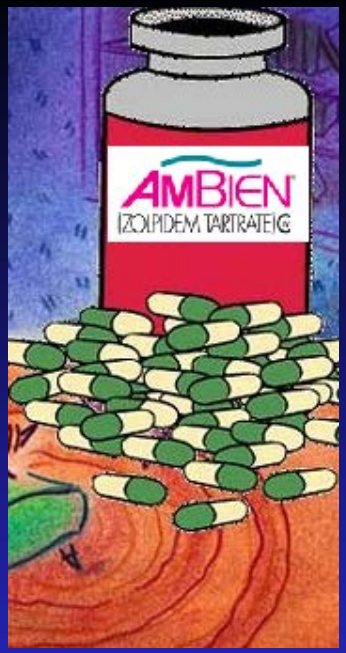
Yamadera et al. 2013 Sleep and Biological Rhythms

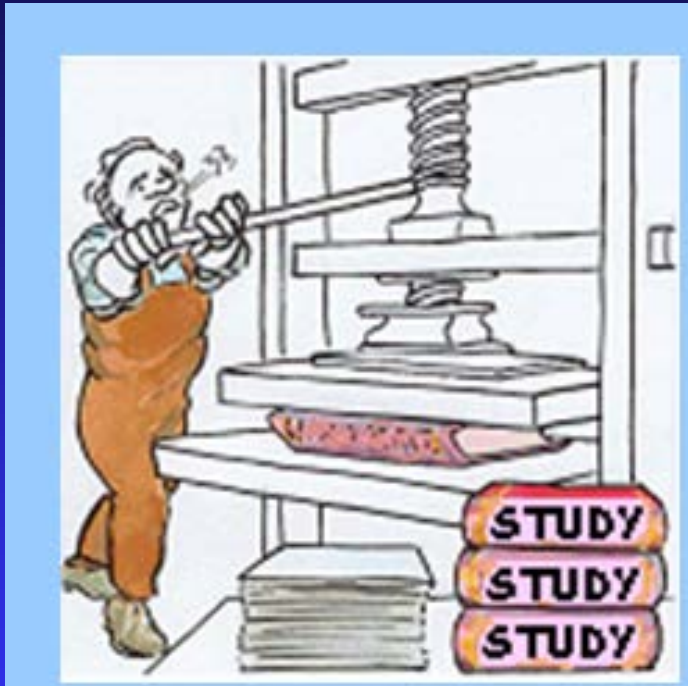


THOUGH WE HAVE SAID IT BEFORE
IT BEARS REPEATING



In an ideal world, the choice of therapy would be based on the following very simple principles: Pharmacotherapy is indicated in the instances where the condition is acute and the need for immediate symptom reduction is the primary consideration. This indication also carries with it the possibility that short term treatment for acute insomnia may have some prophylactic value against the development of chronic insomnia. That is, if sedative hypnotics are more frequently prescribed for such things as jet lag, insomnia related to acute medical illness or insomnia secondary to transient life stressors (e.g., bereavement), such a strategy may prevent the engagement of behavioral strategies which are thought to perpetuate insomnia and lead to conditioned arousal. Behavioral treatment is indicated in the instances where the condition is chronic and/or in acute cases where 1) pharmacotherapy is contraindicated, e.g. in pediatric or geriatric patients, 2) when there is a potential for drug interactions, or 3) when patients present with a history of substance abuse.

Journal of Psychosomatic Research, 54 (1): 51-59, 2003.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FOR A SURPRISINGLY GOOD 30K VIEW OF TX

INSOM

The long-term management of chronic insomnia: recommendations for primary care physicians

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Insomnia is a highly prevalent condition that often becomes chronic and is associated with significant psychiatric and medical morbidity. There are several efficacious strategies for the treatment of insomnia. Historically, however, there has been little advice for the long-term management of this condition. This article reviews the historical context for the treatment strategies currently available, previews in a general way the new therapies in development, and suggests reasonable practice guidelines for both the short- and long-term management of insomnia.

Barbiturates are thought to have negative effects on sleep architecture.

Insomnia is a very common disorder with a prevalence of approximately 25% for acute insomnia and 10% for chronic insomnia. Chronic insomnia is associated with increased fatigue, cognitive impairment, mood disturbance, physical complaints and reduced quality of life.[1–3] Beyond these sequelae, there is now considerable evidence that chronic insomnia increases the risk of substance abuse,[2] psychiatric illness (especially major depressive disorder),[3–11] hypertension and/or cardiovascular disease,[12] dysregulation of glucose homeostasis,[13] immunosuppression[14] and increased mortality.[15] Despite the prevalence and consequences of allowing insomnia to go untreated, there are no best practice guidelines for its long-term management. In this review, we will provide:

- some perspective on how treatment has been conducted in the past
- information on current therapies
- an overview of novel approaches in development
- considerations for the management of chronic insomnia.

Historical context

For several decades beginning in the 1970s, insomnia was considered a 'symptom' not a 'disorder'. To the extent that insomnia was considered 'just a symptom' of medical or psychiatric disease, it was believed that the treatment of the parent disorder was sufficient and would result in the resolution of the insomnia. Long-term management of insomnia, therefore, was thought to be unnecessary.

Despite the 'only a symptom' perspective, targeted treatments were developed and evaluated. Initially barbiturates, then benzodiazepines and, more recently, a class of compounds referred to as benzodiazepine receptor agonists (BZRAs) (also known as 'non-benzodiazepines') were indicated for use as sedative hypnotics. While all three classes have demonstrated efficacy, barbiturates were shown to have a high abuse potential and a low lethal dose profile, and were also found to have what are thought to be negative effects on sleep architecture (reduced amount of rapid eye movement and slow-wave sleep). The benzodiazepine hypnotics were also thought to have similar attributes, but there is little or no evidence for these claims. Benzodiazepines bind to the benzodiazepine receptor component of the γ-aminobutyric acid (GABA) receptor chloride channel complex. The α-subunit is the main receptor site on the GABA complex and at least five α-receptor subtypes have been recognized. Most of the older benzodiazepines bind to multiple α-receptors. This nonselective binding is thought to cause their hypnotic, anticonvulsant, muscle relaxant and other CNS actions.

Despite both the clinical and market success of the barbiturates and the benzodiazepines, BZRAs gained widespread acceptance as the therapeutic standard in practice owing to the fact that this class of medication did not possess the aforementioned

FOR SOMETHING A BIT MORE CONTEMPORARY

CLINICAL PRACTICE

Caren G. Solomon, M.D., M.P.H., *Editor*

Insomnia Disorder

John W. Winkelman, M.D., Ph.D.

This Journal feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author's clinical recommendations.

A 77-year-old overweight woman with hypertension and arthritis reports that she has had trouble sleeping for "as long as I can remember." She has taken hypnotic medications nightly for almost 50 years; her medication was recently switched from lorazepam (1 mg), which had been successful, to trazodone (25 mg) by her primary care physician, who was concerned about her use of the former. She spends 9 hours in bed, from 11 p.m. to 8 a.m. She has only occasional difficulty falling asleep, but she awakens two to three times per night to urinate and lies in bed for over an hour at those times, "just worrying." How should her case be managed?

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N Engl J Med 2015;373:1437-44.
DOI: 10.1056/NEJMcp1412740

An audio version of this article is available at NEJM.org

THE CLINICAL PROBLEM

Dissatisfaction with sleep owing to difficulty falling asleep or staying asleep or to waking up too early is present in roughly one third of adults on a weekly basis.[1] For most, such sleep difficulties are transient or of minor importance. However, prolonged sleeplessness is often associated with substantial distress, impairment in daytime functioning, or both. In such cases, a diagnosis of insomnia disorder is appropriate. Reductions in perceived health[2] and quality of life,[3] increases in workplace injuries and absenteeism,[4] and even fatal injuries[5] are all associated with chronic insomnia. Insomnia symptoms may also be an independent risk factor for suicide attempts and deaths from suicide, independent of depression.[6] Neuropsychological testing reveals deficits in complex cognitive processes, including working memory and attention switching,[7] which are not simply related to impaired alertness.

Older diagnostic systems attempted to distinguish "primary" from "secondary" insomnia on the basis of the inferred original cause of the sleeplessness. However, because causal relationships between different medical and psychiatric disorders and insomnia are often bidirectional, such conclusions are unreliable. In addition, owing to the poor reliability of insomnia subtyping[8] based on phenotype or pathophysiology, the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders*[9] takes a purely descriptive approach that is based on the frequency and duration of symptoms (Table 1), allowing a diagnosis of insomnia disorder independent of, and in addition to, any coexisting psychiatric or medical disorders. The clinician should monitor whether treatment of such coexisting disorders normalizes sleep, and if not, treat the insomnia disorder independently.

COEXISTING CONDITIONS

Insomnia is more common in women than in men, and its prevalence is increased in persons who work irregular shifts and in persons with disabilities.[2] Although

BREAK

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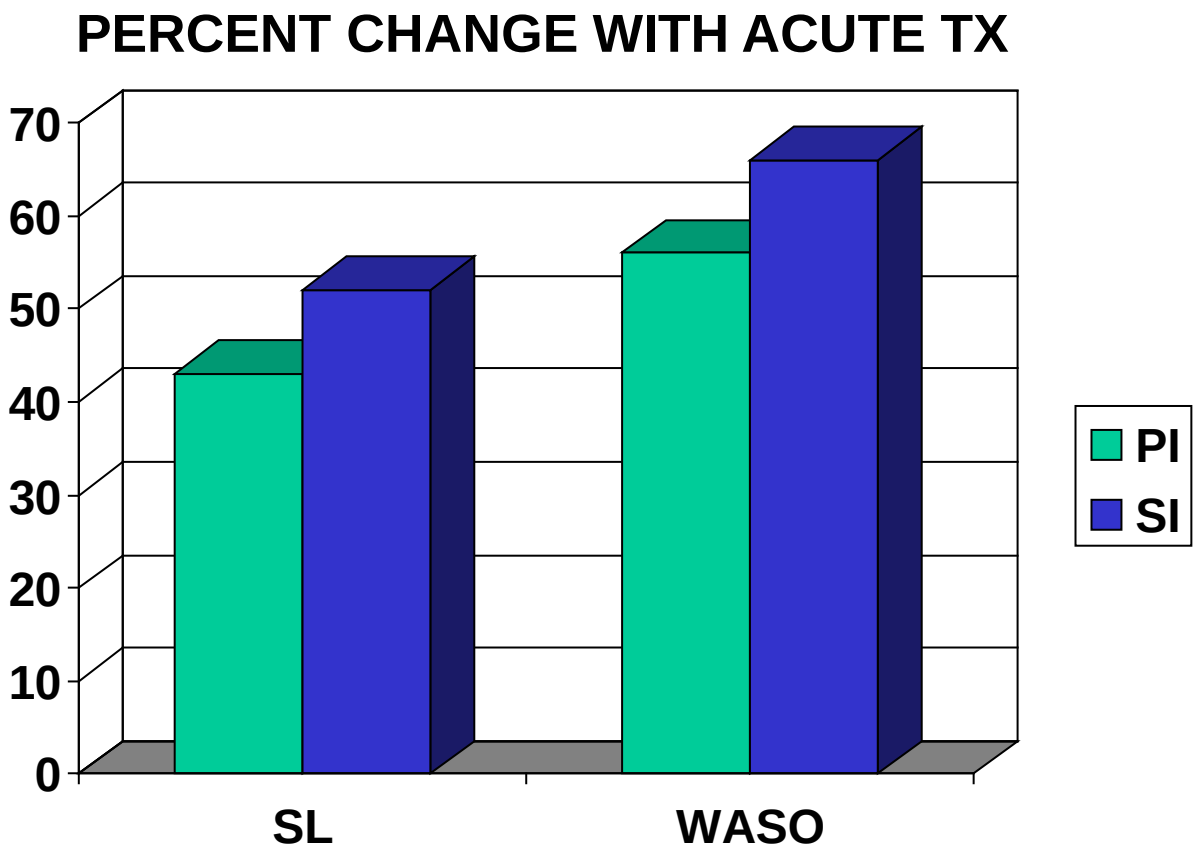
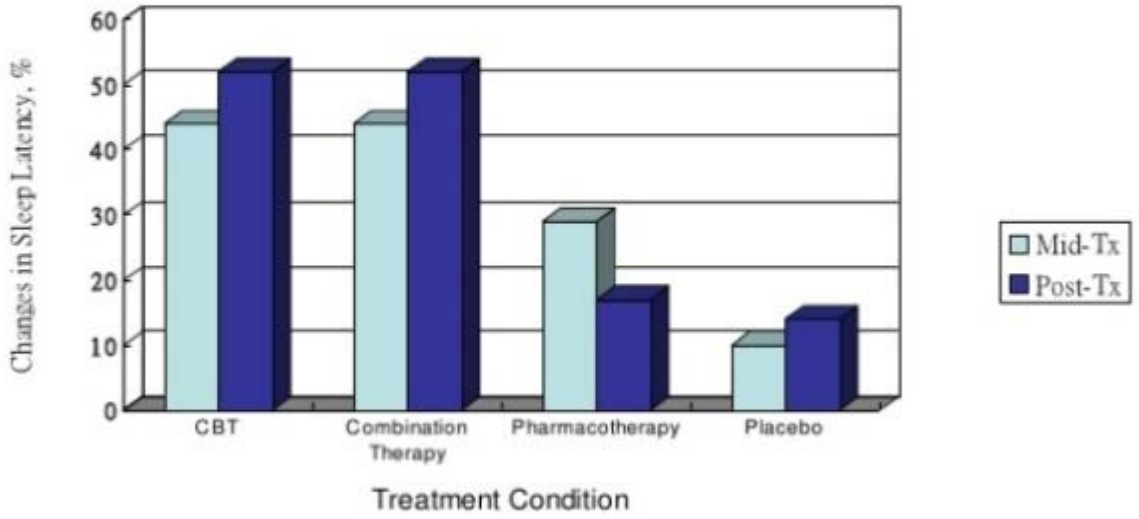


PI: SMITH, PERLIS, ET AL., 2002 SI: WU ET AL. 2015

CBT vs. Hypnotic for Sleep-Onset Insomnia

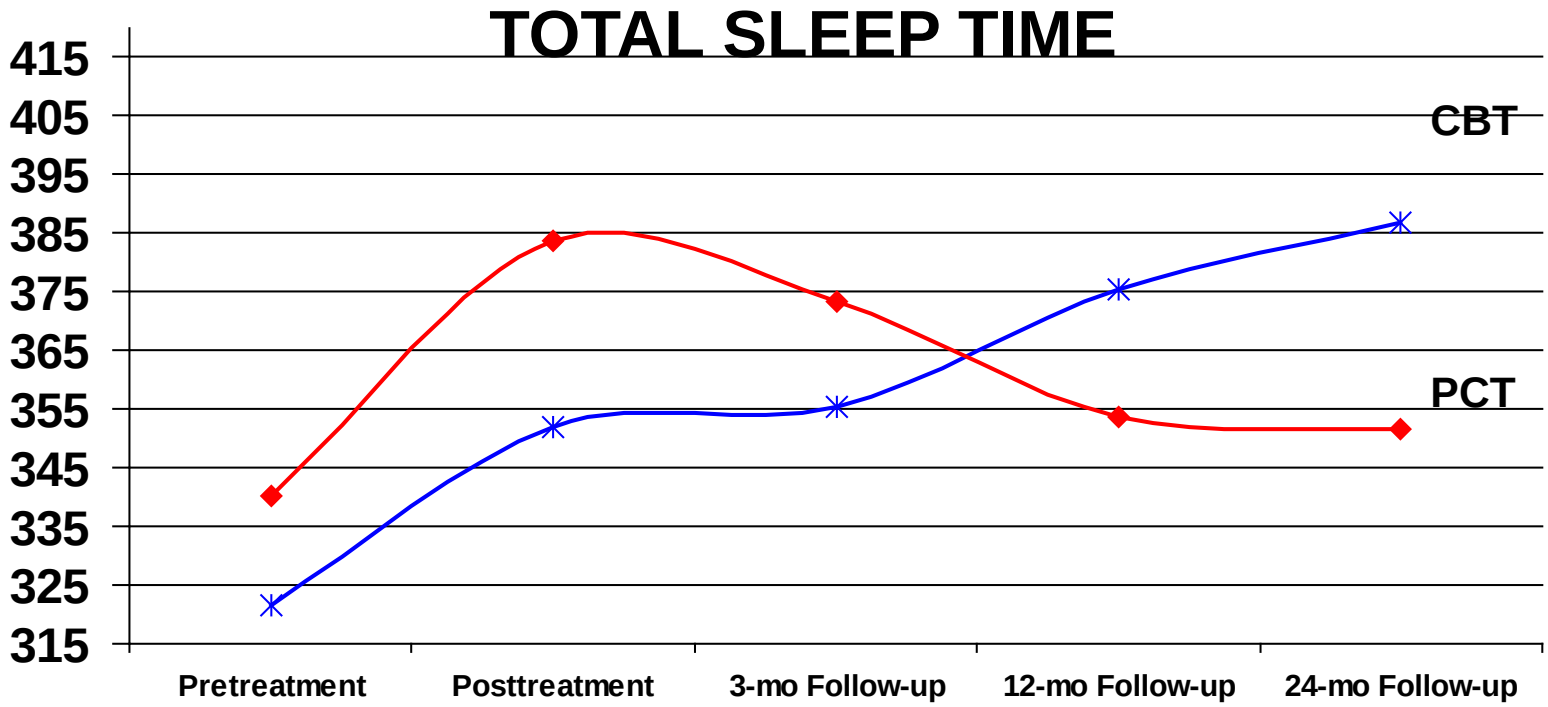


(Jacobs, G.D., et al. Ach Intern Medicine 2004;164: 1888-1896)

SLEEP

Morin 1999 (28) Canada	RCT 6	78 patients, age 55 and up 24 months	Group CBT-I 8 weekly sessions	Temazepam, see comment	Sleep diaries, polysomnography	Dose 7.5 mg→30 mgas needed Study also included placebo and combined therapy groups. Adverse effects reported in (37) Attitudes reported in (38)

NEXT REMOVES INTIAL DIFFERENCE



Morin et al, 1999

Logging on for Better Sleep: RCT of the Effectiveness of Online Treatment for Insomnia

Norah Vincent, PhD; Samantha Lewycky, MA

Department of Clinical Health Psychology, University of Manitoba, Manitoba, Canada

Study Objectives: Despite effective cognitive behavioral treatments for chronic insomnia, such treatments are underutilized.[1,2] This study evaluated the impact of a 5-week, online treatment for insomnia.

Design: This was a randomized controlled trial with online treatment and waiting list control conditions.

Participants: Participants were 118 adults with chronic insomnia.

Setting: Participants received online treatment from their homes.

Intervention: Online treatment consisted of psychoeducation, sleep hygiene, and stimulus control instruction, sleep restriction treatment, relaxation training, cognitive therapy, and help with medication tapering.

Measurement and Results: From pre- to post-treatment, there was a 33% attrition rate, and attrition was related to referral status (i.e., dropouts were more likely to have been referred for treatment rather than recruited from the community). Using a mixed model analysis of variance procedure (ANOVA), results showed that online treatment produced statistically significant improvements in the primary end points of sleep quality, insomnia severity, and daytime fatigue. Online treatment also produced significant changes in process variables of pre-sleep cognitive arousal and dysfunctional beliefs about sleep.

Conclusions: Implications of these findings are that identification of who most benefits from online treatment is a worthy area of future study.

Keywords: Online treatment, insomnia, self-administered treatment

Citation: Vincent N; Lewycky S. Logging on for better sleep: RCT of the effectiveness of online treatment for insomnia. *SLEEP* 2009;32(6):807-815.

CHRONIC INSOMNIA IS A PROBLEM PLAGUING 9% TO 9.5% OF THE POPULATION.[1,2] SUFFERERS EXPERIENCE REGULAR NOCTURNAL PROBLEMS WITH SLEEP AND report associated daytime impairment. Cognitive behavioral and pharmacotherapies have been developed for chronic insomnia and found to produce robust changes in sleep parameters.[3] Research in the area of treatment preference shows that individuals with insomnia tend to prefer behavioral over pharmacological treatments.[4,5] Given that chronic insomnia is a prevalent condition and that individuals are favorably predisposed to behavioral methods to treat this problem, only 5% to 46% seek treatment for their sleep disorder.[1,2,6,7] This rate of treatment seeking is similar to that in the area of mental health,[8] however, relatively little is known about the reasons for failure to seek treatment for insomnia. One exception is Stinson, Tang, and Harvey[9] who surveyed help-seeking and non–help-seeking adults with insomnia regarding their reasons for failing to utilize or delaying their use of treatment for insomnia. Participants could report more than one reason. Of this sample, 57% reported a belief that poor sleep would resolve on its own and/or one should be able to manage insomnia independently, 38% indicated that there was a lack of awareness of available treatment options, 31% noted a perception of treatment as ineffective or unattractive, 17% referred to a stigma surrounding insomnia, and 11% endorsed personal constraints regarding treatment-seeking. Other surveys have found that the most frequent reasons given for not consulting about mental health problems are the beliefs that these problems will go away by themselves and that individuals can manage on their own.[10] Some of the noted impediments to help-seeking could potentially be addressed through the provision of self-administered treatment.

Submitted for publication November, 2008
Submitted in final revised form February, 2009
Accepted for publication February, 2009
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Self-Administered Treatments for Insomnia

A recent review of self-help treatments for insomnia showed that there have been a number of published outcome studies in this area.[11-18] In these studies, treatment has been delivered using manuals, audiotapes, television, video, telephone consultation, and the Internet. Currie[19] reviewed the outcomes of these studies, which mainly used media-recruited individuals, and concluded that outcomes from self-help approaches were positive but less favorable than those from in-person psychological treatment. In these investigations, the degree to which self-help treatments were delivered as intended was unclear, as none of the studies assessed how adherent participants were to self-administered treatment with the exception of Mimeault and Morin.[20] Unfortunately these authors did not report on the actual frequency of adherence but did note that treated individuals were similar to controls in terms of self-reported adherence. One of the most promising self-administered approaches with the potential to reach a large number of people is Internet-based treatment. Although there have been a number of Internet-based treatments for other health problems, the only published study of such treatments for insomnia was conducted by Strom and colleagues.[18] Strom et al. developed a 5-week Swedish online treatment for insomnia and evaluated it with 109 community-recruited individuals diagnosed with DSM-IV chronic primary insomnia. A number of interesting results emerged from this study including the finding that the treatment produced changes in sleep parameters for primary study variables, and that the rate of attrition (24%) was comparable to North American in-person psychotherapy standards (22%).[21]

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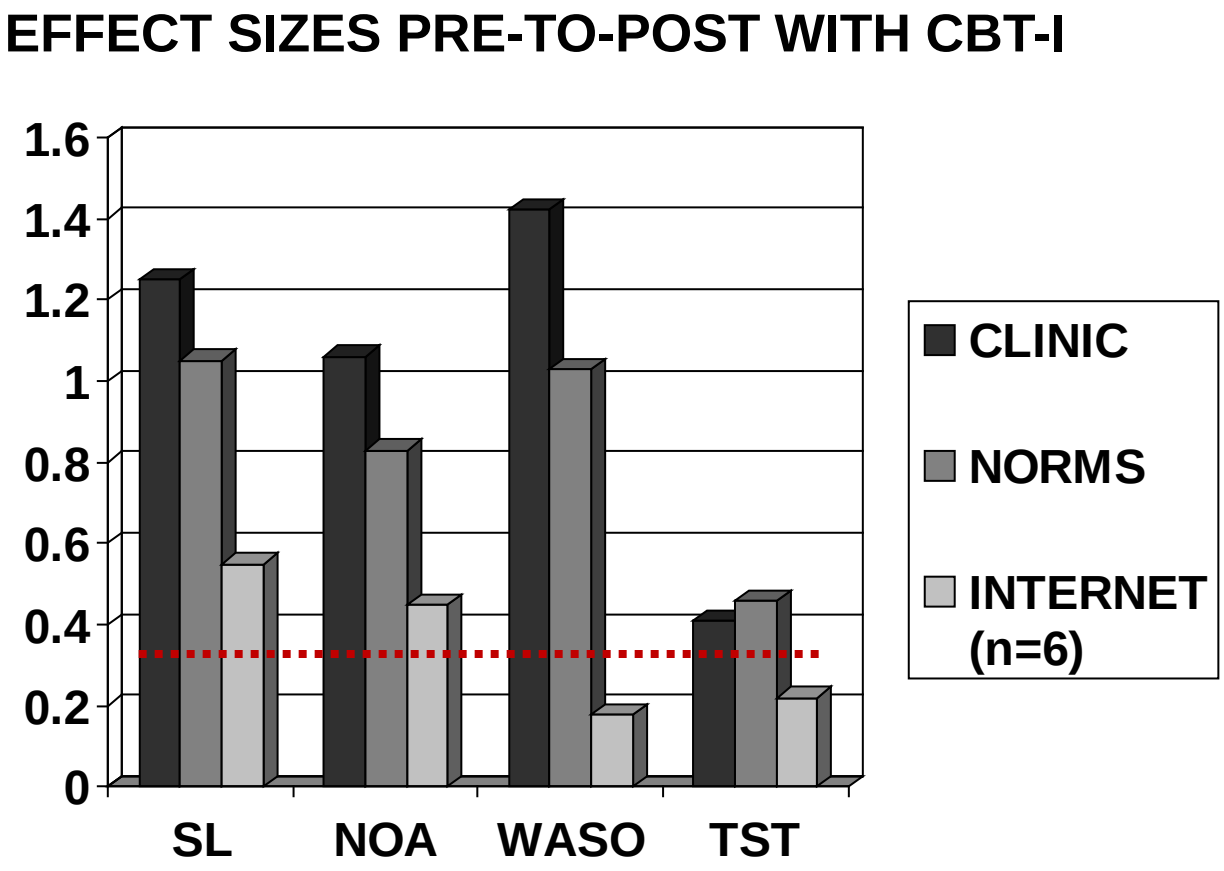

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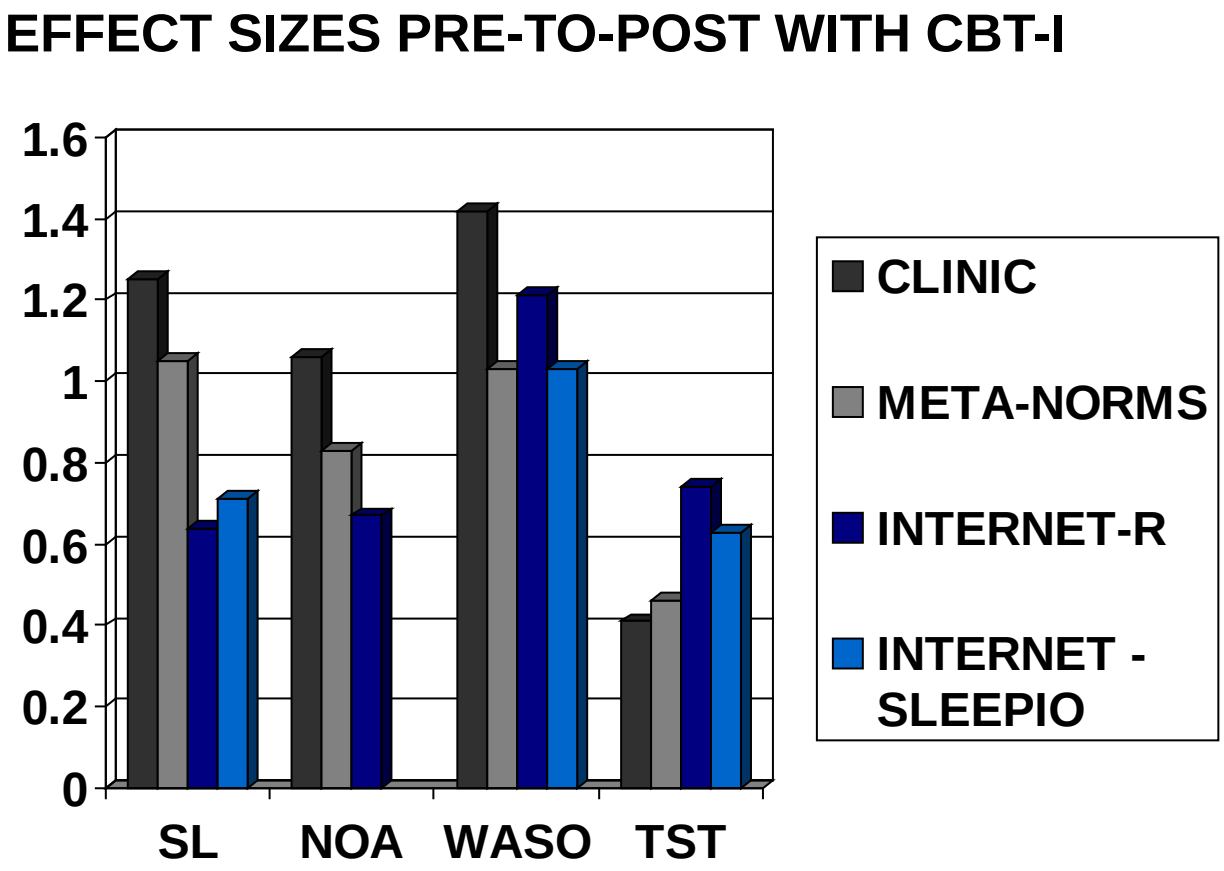

Arch Gen Psychiatry. 2009 Jul;66(7):692-8

SUMMARY
"NOW AND ZEN"

	NIH – 1983	NIH – 2005
Definition		
Treatment		
Other		

PHARMACOTHERAPY

SOME HUMOR RE: PHARMACOTHERAPY
BEFORE WE BEGIN