ETIOLOGY OF INSOMNIA & TREATMENT IMPLICATIONS
WHO NEEDS A MODEL OF INSOMNIA?

“The only problem with insomniacs is they don’t get enough sleep”
IT’S THAT SIMPLE
AND
IT’S NOT THAT SIMPLE
HOW DOES THIS CONDITION DEVELOP?
WHAT IS IT?
WHAT IS THE ETIOLOGY OF INSOMNIA?

UNKNOWN
WHAT IS THE PATHOPHYSIOLOGY OF INSOMNIA?

UNKNOWN
ANY IDEAS ABOUT WHAT INSOMNIA IS AND HOW IT DEVELOPS?
ACTUALLY THERE ARE MORE THAN A FEW
Etiology and Pathophysiology of Insomnia
Michael Lloyd Perls; Jason Gordon Elts; Jacqueline DeMichele Kloss; Dieter Riemann

Chapter 82

Etiology and Pathophysiology of Insomnia

ABSTRACT

Of all the sleep disorders, insomnia is perhaps the one on which there has been a substantial amount of research over the years. This may be in part because a homework is required for comprehension of the impact of limbic and autonomic nervous system abnormalities on sleep. In this chapter, we review models of the etiology and pathophysiology of insomnia and examine the role of the autonomic nervous system in the disturbances. We discuss the current understanding of the role of the autonomic nervous system in the pathophysiology of insomnia and the role of sleep disorders in the treatment of insomnia.

Insomnia is often considered a disorder of the autonomic nervous system, as it is the case in primary insomnia, whereas it is also associated with many other medical conditions. This chapter will cover the following topics:

- The role of the autonomic nervous system in the etiology and pathophysiology of insomnia
- The role of sleep disorders in the treatment of insomnia
- The role of sleep disorders in the treatment of insomnia

Chapter Highlights

- Since 1990, there has been a proliferation of theoretical perspectives on the etiology and pathophysiology of insomnia that includes nine major models. The centered concepts for each of these models include the following:
  - Sleep-disruption theories
  - Stimulus control theories
  - Cognitive-behavioral theories
  - Biological theories
- The role of the autonomic nervous system in the etiology and pathophysiology of insomnia
- The role of sleep disorders in the treatment of insomnia

APPENDIX

Appendix A: The Role of the Autonomic Nervous System in the Etiology and Pathophysiology of Insomnia

Appendix B: The Role of Sleep Disorders in the Treatment of Insomnia

Pathways and Weaknesses

The success of the approach is often determined by the patient's ability to engage in the therapy. The patient must have a complete understanding of the therapy's role in the treatment of insomnia.

REFERENCES


The following references provide additional information on the etiology and pathophysiology of insomnia:


THE PHYSIOLOGIC PERSPECTIVE
THE PHYSIOLOGIC MODEL

Hyperarousal → Insomnia
WHAT IS HYPERAROUSAL?

DO PATIENTS WITH INSOMNIA EXHIBIT THIS?
WHAT IS HYPERAROUSAL?

A LEVEL OF PHYSIOLOGIC AROUSAL THAT INTERFERES WITH THE INITIATION AND MAINTENANCE OF SLEEP

CLASSICAL MEASURES

• HEART RATE (HR)
• RESPIRATION RATE (RR)
• MUSCLE TONUS (EMG)
• TEMPERATURE (CBT)
• STARTLE RESPONSE (GSR)
DO INSOMNIA PATIENTS EXHIBIT INCREASED PHYSIOLOGIC AROUSAL?
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Age (PS and GS)</td>
<td>25/26</td>
<td>18/18</td>
<td>19/19</td>
<td>31/27</td>
<td>51/51</td>
<td>34/34</td>
</tr>
<tr>
<td>Sample Size (PS and GS)</td>
<td>16/16</td>
<td>??/??</td>
<td>10/11</td>
<td>12/12</td>
<td>18/18</td>
<td>24/25</td>
</tr>
<tr>
<td>Recruitment (indicated Insomnia Research)</td>
<td>Yes</td>
<td>No</td>
<td>??</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Medical Screening</td>
<td>??</td>
<td>??</td>
<td>??</td>
<td>Yes</td>
<td>??</td>
<td>Yes</td>
</tr>
<tr>
<td>Psych Screen</td>
<td>??</td>
<td>??</td>
<td>??</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Sleep Dx Screen</td>
<td>??</td>
<td>??</td>
<td>??</td>
<td>Yes</td>
<td>??</td>
<td>Yes</td>
</tr>
<tr>
<td>Insomnia Complaint (for the PS)</td>
<td>No</td>
<td>??</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>PS5 study</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>PS5 Confirmed Insomnia</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Measures</th>
<th>During the Day</th>
<th>During Sleep</th>
<th>During Sleep</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Heart Rate - Prior to Sleep Onset</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Heart rate - During Sleep</td>
<td>↑</td>
<td>ns</td>
<td>↑</td>
</tr>
<tr>
<td>Respiration Rate - During the Day</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Respiration Rate - Prior to Sleep Onset</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Respiration Rate - During Sleep</td>
<td>↑</td>
<td>ns</td>
<td>↑</td>
</tr>
<tr>
<td>Temperature°- During the Day</td>
<td>↑</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature - Prior to Sleep Onset</td>
<td>↑</td>
<td>ns</td>
<td>↑</td>
</tr>
<tr>
<td>Temperature - During Sleep</td>
<td>↑</td>
<td>ns</td>
<td>↑</td>
</tr>
<tr>
<td>Muscle Tension - During the Day</td>
<td>↑</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muscle Tension - Prior to Sleep Onset</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Muscle Tension - During Sleep</td>
<td>↑</td>
<td>ns</td>
<td>↑</td>
</tr>
<tr>
<td>Skin Resistance - During the Day</td>
<td>↑</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin Resistance - Prior to Sleep Onset</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Skin Resistance - During Sleep</td>
<td>↑</td>
<td>ns</td>
<td>↑</td>
</tr>
<tr>
<td>Peripheral Vasoconstrictivity - During the Day</td>
<td>↑</td>
<td>ns</td>
<td>↑</td>
</tr>
<tr>
<td>Peripheral Vasoconstrictivity - Prior to Sleep Onset</td>
<td>↑</td>
<td>ns</td>
<td>↑</td>
</tr>
<tr>
<td>Peripheral Vasoconstrictivity - During Sleep</td>
<td>↑</td>
<td>ns</td>
<td>↑</td>
</tr>
</tbody>
</table>
WHAT IS HYPERAROUSAL?

A LEVEL OF PHYSIOLOGIC AROUSAL THAT INTERFERES WITH THE INITIATION AND MAINTENANCE OF SLEEP

CONTEMPORARY MEASURES

• HEART RATE VARIABILITY (HRV)
• METABOLIC RATE
• CORTISOL LEVEL
HRV

METABOLIC RATE

HPA AXIS ABNORMALITIES

Fig. 2. Twenty-four-hour plasma cortisol concentrations in insomniacs (■) and controls (○). The thick black line indicates the sleep recording period. The error bar indicates SE. *P < 0.01.

Vgontzas et al. 2001. Journal of Clinical Endocrinology & Metabolism
Figure 2: Twenty-four-hour plasma cortisol concentrations in insomniacs and controls. The thick black line indicates the sleep recording period. The error bar indicates the standard error of the mean.
Q: IS THE LEVEL OF AROUSAL ENOUGH TO INTERFERE WITH SLEEP INITIATION OR MAINTENANCE?

DOES THE AROUSAL LEVEL COMPARE TO THIS?!
IT’S DOUBTFUL
DOES THE MODEL EXPLAIN HOW THE HYPERAROUSAL CONDITION COMES INTO EXISTENCE?
DOES THE MODEL EXPLAIN HOW ACUTE INSOMNIA BECOMES CHRONIC AND HOW THE CONDITIONS DIFFER?
CAN THIS MODEL EXPLAIN THE VARIOUS INSOMNIA PHENOTYPES (TYPES AND SUBTYPES)

PRIMARY INSOMNIA / INSOMNIA DISORDER
- PSYCHOPHYSIOLOGIC INSOMNIA
- IDIOPATHIC INSOMNIA
- PARADOXICAL INSOMNIA
- SLEEP HYGIENE DISORDER
- PHYSIOLOGIC INSOMNIA
- INSOMNIA NOS

SLEEP COMPLAINT
One or more of the symptoms below
- Difficulty Falling Asleep
- Difficulty Staying Asleep
- Waking Too Early
  - Initial Insomnia
  - Middle Insomnia
  - Late Insomnia
CLINICAL REVIEW

The hyperarousal model of insomnia: A review of the concept and its evidence

Dieter Kriemann, Kai Spiegelhalder, Bernd Feige, Ulrich Voderholzer, Matthias Berger, Michael Perlis, Christoph Nissen

SUMMARY

Primary insomnia is defined as difficulties in falling asleep, maintaining sleep or non-restorative sleep accompanied by significantly impaired daytime functioning in the absence of a specific physical, mental or substance-related cause. The current review provides substantial support for the concept that hyperarousal processes from the molecular to the higher level system play a key role in the pathophysiology of primary insomnia. Autonomic, neuroendocrine, neuroimmunological, electrophysiological and neuromaging studies demonstrate increased levels of arousal in primary insomnia during both night and daytime. In the light of immunological theories of sleep-wake regulation, primary insomnia may be conceptualized as a final common pathway resulting from the interplay between a genetic vulnerability for an imbalance between arousal and sleep-inducing brain activity, psychological adversal stressors and perpetuating mechanisms including dysfunctional sleep-related behavior, learned sleep preventing associations and other cognitive factors like tendency to worry/minimize.

Introduction

Insomnia as a diagnostic entity is defined as a complaint of prolonged sleep latency, difficulties in maintaining sleep, the experience of non-refreshing or poor sleep coupled with impairments of daytime functioning, including reduced alertness, fatigue, exhaustion, dysphoria and other symptoms. The complaints have to endure for at least 4 weeks to be diagnosed as insomnia. The Diagnostic and Statistical Manual of the American Psychiatric Association (DSM-I) classifies insomnias into primary insomnia (PI), insomnia related to a medical or mental disease and insomnia related to the intake or abuse dependency from substances. The International Classification of Sleep Disorders (ICSD) goes beyond that approach and specifies 11 insomnia subtypes encompassing among others acute, psychophysiological, parasomnias, idiopathic and substance-induced insomnia.

Insomnia as a symptom is a highly prevalent health complaint affecting up to 50% of the general population depending on criteria applied. Estimates for the prevalence of PI as a diagnostic entity in the general population range from 3 to 5%. Research diagnostic criteria for insomnia now provide operationalized and standardized criteria for the diagnosis of insomnia and its subtypes.

Polysomnographic research on insomnia revealed a remarkable discrepancy between the subjective experience of insomnia and polysomnographically rather and disturbed sleep in many patients with primary insomnia. Thus, polysomnography (PSG), in contrast to other fields of clinical sleep medicine, has not become the gold standard for the diagnosis of insomnia. Insomnia diagnosis and assessment is based on subjective reports (sleep questionnaires) of sleep behavior and relies on sleep diaries filled out every evening and morning (for an overview of relevant instruments see ).

The effectiveness of cognitive-behavioral treatment for insomnia (CBT-I) compared to the risks inherent with pharmacological insomnia treatment (e.g., benzodiazepines) may have added to the conceptualization of PI as primarily a psychological disorder and negligence to study its biological aspects (comparably to other sleep disorders or other disorders in the field of mental health).

The "hyperarousal" perspective of insomnia has gained widespread attention as an integrative approach to the pathophysiology of insomnia (especially primary insomnia (PI) or psychophysiological
TARGETS FOR TREATMENT
PHYSIOLOGIC MODEL OF INSOMNIA (GENERAL)

THE PHYSIOLOGIC MODEL

Hyperarousal \rightarrow Insomnia

RELAXATION
HYPNOTICS
ANXIOLYTICS
MUSCLE RELAXANTS

ACTUALLY SRT !
THE COGNITIVE PERSPECTIVE
COGNITIVE MODEL OF INSOMNIA
(GENERAL)

THE COGNITIVE MODEL

- Problem solving
- Rumination and worry

→ Insomnia
INSOMNIA OCCURS AS A RESULT OF WORRY
WORRY – CLASSIC
WORRY – CONTEMPORARY
DOES CHRONIC INSOMNIA OCCUR BECAUSE OF

WORRY
RUMINATION
INTRUSIVE THOUGHTS

SELECTIVE ATTENTION
SLEEP-RELATED INTENTION AND EFFORT
MAYBE

OR MAYBE THE COGNITIVE FACTORS ARE “WIND TO THE FLAME”

THAT IS, COGNITIVE FACTORS SERVE TO MAKE THE INSOMNIA MORE SEVERE AND MORE CHRONIC
CONSIDER THIS:

IN THE CASE OF **CHRONIC INSOMNIA**

IS IT THE CASE THAT WORRY KEEPS ONE AWAKE

OR

**THAT ONE WORRIES BECAUSE ONE IS AWAKE?**
DOES THE MODEL EXPLAIN HOW THE HYPERAROUSAL CONDITION COMES INTO EXISTENCE?
DOES THE MODEL EXPLAIN HOW ACUTE INSOMNIA BECOMES CHRONIC AND HOW THE CONDITIONS DIFFER?

Diagram:
- GOOD SLEEP → WORRY → HYPER AROUSAL → ACUTE INSOMNIA → WORRY → GOOD SLEEP
- ACUTE INSOMNIA → WORRY → CHRONIC INSOMNIA
CAN THIS MODEL EXPLAIN THE VARIOUS INSOMNIA PHENOTYPES (TYPES AND SUBTYPES)

PRIMARY INSOMNIA / INSOMNIA DISORDER

PSYCHOPHYSIOLOGIC INSOMNIA
IDIOPATHIC INSOMNIA
PARADOXICAL INSOMNIA
SLEEP HYGIENE DISORDER
PHYSIOLOGIC INSOMNIA
INSOMNIA NOS

SLEEP COMPLAINT
One or more of the symptoms below

- Difficulty Falling Asleep
- Difficulty Staying Asleep
- Waking Too Early
- Initial Insomnia
- Middle Insomnia
- Late Insomnia
COGNITIVE MODEL OF INSOMNIA (GENERAL)

THE COGNITIVE MODEL

Problem solving
Rumination and worry

Insomnia

COGNITIVE THERAPY
HYPNOTICS
MBSR
GEN. PSYCHOTHERAPY
ANXIOLYTICS
DOPAMINE ANTAGONISM
AYTPICAL ANTIPSYCHOTICS

ACTUALLY SRT & SCT
THE BEHAVIORAL PERSPECTIVE
THE SPIELMAN MODEL
(AKA 3 FACTOR OR 3P MODEL)

“The best cure for insomnia is to get a lot of sleep”

-- W.C. Fields
DOES THE MODEL EXPLAIN HOW THE HYPERAROUSAL CONDITION COMES INTO EXISTENCE?

YES.
DOES THE MODEL EXPLAIN HOW ACUTE INSOMNIA BECOMES CHRONIC AND HOW THE CONDITIONS DIFFER?

YES.
3rd P – SLEEP EXTENSION

HOW TIME IN BED VARIES WITH INSOMNIA

HOW SLEEP OPPORTUNITY IS EXPANDED TO RECOVER LOST SLEEP
SO... IF SLEEP EXTENSION IS THE PROBLEM
THEN SLEEP RESTRICTION IS THE SOLUTION
Is this true?
SO FAR...
CAN THIS MODEL EXPLAIN THE VARIOUS INSOMNIA PHENOTYPES (TYPES AND SUBTYPES)

PRIMARY INSOMNIA / INSOMNIA DISORDER
PSYCHOPHYSIOLOGIC INSOMNIA
IDIOPATHIC INSOMNIA
PARADOXICAL INSOMNIA
SLEEP HYGIENE DISORDER
PHYSIOLOGIC INSOMNIA
INSOMNIA NOS

SLEEP COMPLAINT
One or more of the symptoms below

- Difficulty Falling Asleep
- Difficulty Staying Asleep
- Waking Too Early
  - Initial Insomnia
  - Middle Insomnia
  - Late Insomnia
PROBABLY NOT
DOES CHRONIC INSOMNIA OCCUR SOLELY IN RELATION TO SLEEP EXTENSION?

Schematic representation by Michael Smith PhD
PROBABLY NOT
ASSUMING TX (CBT-I) ENTIRELY ELIMINATES THE BEHAVIORS THAT PERPETUATE INSOMNIA

WHY ARE PATIENTS NOT CURED?
AVERAGE RESPONSE = ~ 50%

IS THERE SOMETHING MISSING FROM THE BEHAVIORAL MODEL?
THE BEHAVIORAL MODEL FOCUSES ON INSTRUMENTAL AND NOT CLASSICAL CONDITIONING
CLASSICAL CONDITIONING

NORMAL SITUATION
BEDROOM/BEDTIME → SLEEPINESS & SLEEP

ACUTE INSOMNIA SITUATION
BEDROOM/BEDTIME + LIFE STRESS INDUCED SOMATIC AROUSAL → SCD
BEDROOM/BEDTIME + LIFE STRESS INDUCED CORTICAL AROUSAL → SCD

CHRONIC INSOMNIA SITUATION
BEDROOM/BEDTIME + LIFE STRESS INDUCED SOMATIC AROUSAL → SCD
BEDROOM/BEDTIME + LIFE STRESS INDUCED CORTICAL AROUSAL → SCD

PATIENT’S TELL YOU ABOUT THIS ALL THE TIME!
SO IF ONE TAKES INTO ACCOUNT CONDITIONING

THE THREE FACTOR MODEL COULD BE REPRESENTED AS A FOUR FACTOR MODEL
THE FOUR FACTOR MODEL

4 FACTOR MODEL

- Conditioning (Pavlovian)
- Perpetuating
- Precipitating
- Predisposing

Threshold

Pre-Morbid  Acute  Early  Chronic  Acute Tx  + Response

Perlis, Pigeon and Smith; Principles and Practice of Sleep Medicine Chapter 60
DOES CHRONIC INSOMNIA OCCUR SOLELY IN RELATION TO PHYSIOLOGIC, COGNITIVE, AND BEHAVIORAL FACTORS?
PROBABLY NOT
IT’S LIKELY THAT MODERATORS & MEDIATORS ARE AT PLAY

Figure 2

IV -> independent variable
DV -> dependent (response) variable
MV -> mediator variable
WHAT’S MISSING?
Sleep Homeostasis and Models of Sleep Regulation

Alexander A. Borbély
Peter Achermann

The level of electrophysiological (EEG) slow wave activity (SWA) is determined by the duration of prior sleep and waking. SWA is a marker of neuronal activity (NEEM) sleep intensity and may serve as an indicator of NEEM sleep homeostasis. Power in the range of sleep spindles (spindle frequency activity; SFA) shows an inverse relationship to SWA. This relationship can be accounted for by neuropsychological data. Thalamocortical neurons exhibit oscillations in the range of sleep spindles at an intermediate level of hypocapnia (corresponding to superficial NEEM sleep), and slow oscillations at a high level of hypocapnia (corresponding to deep NEEM sleep). Although the homeostatic NEEM sleep process is largely independent of circadian factors, it interacts with the circadian rhythm of sleep propensity.

The two-process model of sleep regulation is based on the homeostatic process S and the circadian process C. The model can simulate the SWA patterns for a variety of experimental schedules. Essential aspects of the model have been validated by results from forced desynchrony protocols. Other models include the two-oscillator model, the neuronal-oscillation model, and combined models. The incorporation of rapid eye movement (REM) sleep homeostasis is still at an early stage.

There is recent evidence for a local, non-dependant factor of sleep regulation. This concept is derived from autonomic and sleep experiments in non-human primates, and from studies relating specific regional effects in the sleep EEG to brain waves. The modeling approach could be extended to brain sleep.

There are basic components underlying sleep regulation: (1) a homeostatic process determined by sleep and waking, (2) a circadian process, a clock-like mechanism determining the alternation of periods with high and low sleep propensity and being basically independent of sleep and waking, and (3) an ultradian process occurring within sleep and represented by the alternation of the two basic sleep states—non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. This chapter focuses on "sleep homeostasis." Homeostasis has been defined as "the coordinated physiological process which maintains most of the steady states in the organism." The term sleep homeostasis refers to the sleep-wake-dependent aspect of sleep regulation, as homeostatic mechanisms correct deviations from an average "reference" level of sleep. They maintain sleep propensity, which sleep is considered as driven by changes in sleep propensity in response to excess sleep.

The interest in modeling the processes underlying sleep regulation has increased over the past decade. In the research briefing report of the Institute of Medicine, a panel of leading North American experts in basic sleep research recommended that "the homeostatic and circadian influences need to be integrated into a single functional model that can describe both the timing of sleep and its quality." Models help delineate the processes involved in the regulation of sleep and thereby offer a conceptual framework for the analysis of existing and new data.

HOMEOSTATIC REGULATION OF SLEEP

Electrophysiological Slow Wave Activity: A Physiological Indicator of NREM Sleep Homeostasis

Slow-Wave Sleep and Slow Wave Activity: NREM sleep is not a homogenous substrate of sleep, but can be subdivided according to the predominance of electrophysiological (EEG) slow wave activity (SWA). The percentage of slow waves (frequency, 0 to 3 Hz; minimum peak-to-peak voltage, 75 μV²) is the major criterion for scoring human NREM sleep into the stages 2, 3, or 4. Stages 3 and 4 are commonly referred to as slow-wave sleep (SWS). However, the conventional sleep scoring method is inadequate for a quantitative analysis, because the sleep stages are based on either general and arbitrary criteria. Presently, EEG parameters can be assessed by computer-aided methods of signal analysis. One of the most important functional EEG parameters will be referred to as "slow wave activity." It is equivalent to "delta activity" and encompasses components of the EEG signal in the frequency range of slow waves.
BORBELY’S 2 PROCESS MODEL OF NORMAL SLEEP
WHAT ABOUT INSOMNIA?
THE TWO PROCESS MODEL HELP ACCOUNT FOR INSOMNIA SUBTYPE

INITIAL AND LATE INSOMNIA MAY OCCUR WITH SUBTLE PHASE SHIFTS OR SLEEPING OUT OF ONE’S PREFERRED SLEEP PHASE

INITIAL, MIDDLE, OR LATE, MAY OCCUR AS SLEEP HOMEOSTASIS DYSREGULATION (DEPRIME OR EXCESSIVE OPPORTUNITY)
THE TWO PROCESS MODEL HELPS ACCOUNT FOR WHY SLEEP EXTENSION IS A PROBLEM AND WHY SLEEP RESTRICTION WORKS

“IF SLEEP EXTENSION IS THE PROBLEM, SLEEP RESTRICTION IS THE SOLUTION”
TARGETS FOR TREATMENT
FOUR FACTOR MODEL

4 FACTOR MODEL

Threshold

Conditioned Arousal
Perpetuating
Precipitating
Predisposing

Pre-Morbid Acute Early Chronic Acute Tx + Response

STIMULUS CONTROL INST
HYPNOTICS
SADs
OREXIN ANTAGONISM

SLEEP RESTRICTION
STIMULUS CONTROL INST

EXERCISE
RELAXATION
GEN. PSYCHOTHERAPY
SO THESE ARE THE BASIC MODELS
THERE ARE OTHER MODELS WORTH STUDYING DOWN THE ROAD

THE LUNDH MODEL
THE NEUROCOGNITIVE MODEL
THE HARVEY MODEL
THE PSYCHOBIOLOGICAL INHIBITION MODEL
THE NEUROBIOLOGICAL MODEL
THE DROSOPHILA MODEL
THE RODENT MODEL
THE PARALLEL PROCESS MODEL
FROM A CLINICAL POINT OF VIEW
FROM A RESEARCH POINT OF VIEW
“No matter how important sleep may be, it was adaptively deferred when the mountain lion entered the cave.”

SPIELMAN ET AL. 1991
Thank you Jay!
WE LIVE WITH INSOMNIA TODAY BECAUSE, AT SOME POINT, IN OUR EVOLUTIONARY HISTORY INSOMNIA ALLOWED US TO LIVE’

DEAN HANDELEY
SEPRACOR
CIRCA 2005
DINNER
GIVEN THE TWO PROCESS MODEL
WHAT SHOULD BE THE KEY QUESTIONS OF RELEVANCE FOR TX

1. HOW LONG IS THE INDIVIDUAL AWAKE DURING THE DAY?
2. DOES THE INDIVIDUAL NAP (AND WHEN)?
3. WHAT TIME IS THE INDIVIDUAL GOING TO BED?
4. WHAT TIME IS THE INDIVIDUAL GETTING OUT OF BED?