ETIOLOGY AND PATHOPHYSIOLOGY OF INSOMNIA & TREATMENT IMPLICATIONS

SO WHO NEEDS A MODEL?
“The only problem with insomniacs is they don’t get enough sleep”

REVIEW OF INSOMNIA MODELS
RE: ETIOLOGY AND PATHOLOGY
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?

THE PHYSIOLOGIC PERSPECTIVE
WHAT IS HYPERAROUSAL?

Do patients with insomnia exhibit this?

A level of physiologic arousal that interferes with the initiation and maintenance of sleep.

Classical measures:
- Heart rate (HR)
- Respiration rate (RR)
- Muscle tone (EMG)
- Temperature (CRT)
- Startle response (GSR)
DO INSOMNIA PATIENTS EXHIBIT INCREASED PHYSIOLOGIC AROUSAL?

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

Vgontaz et al. 2001. Journal of Clinical Endocrinology & Metabolism

QUESTION: WHY IS THE EFFECT LOCALIZED 20:00-02:00 ?
WHAT CONFOUND MIGHT THERE BE ?
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)

FOR A GOOD REVIEW OF THE EVIDENCE
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
SAFETY BEHAVIORS
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?

CAN THIS MODEL EXPLAIN THE VARIOUS INSOMNIA PHENOTYPES (TYPES AND SUBTYPES)?

THE BEHAVIORAL PERSPECTIVE
THE SPIELMAN MODEL
(AKA 3 FACTOR MODEL)


“The best cure for insomnia is to get a lot of sleep”
-- W.C. Fields
SPIELMAN’S NEW MODEL

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.
CAN THIS MODEL EXPLAIN THE VARIOUS INSOMNIA PHENOTYPES (TYPES AND SUBTYPES)

PROBABLY NOT

DOES CHRONIC INSOMNIA OCCUR SOLELY IN RELATION TO SLEEP EXTENSION?
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(S) ?

THE BEHAVIORAL MODELS FOCUS 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
SO IF ONE TAKES INTO ACCOUNT CONDITIONING

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

THE FOUR FACTOR MODEL

DOES CHRONIC INSOMNIA OCCUR SOLELY IN RELATION TO PHYSIOLOGIC, COGNITIVE, AND BEHAVIORAL FACTORS?
PROBABLY NOT

IT’S LIKELY THAT MODERATORS & MEDIATORS ARE AT PLAY

WHAT’S MISSING?
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?

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 HELP 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

PHYSIOLOGIC MODEL OF INSOMNIA (GENERAL)

THE PHYSIOLOGIC MODEL

Hyperarousal → Insomnia

HYPNOTICS
RELAXATION
ANXIOLYTICS
MUSCLE RELAXANTS

COGNITIVE MODEL OF INSOMNIA (GENERAL)

THE COGNITIVE MODEL

Problem solving
Rumination and worry → Insomnia

HYPNOTICS
GEN. PSYCHOTHERAPY
COGNITIVE THERAPY
MUSK
ANXIOLYTICS
DOPAMINE ANTAGONISM
ATYPICAL ANTI-Psychotics
SO THESE ARE THE BASIC MODELS

THERE ARE OTHER MODELS WORTH STUDYING DOWN THE ROAD

THE LUNDH MODEL
THE NEUROCOGNITIVE MODEL
THE DROSOPHILA MODEL
THE HARVEY MODEL
THE PSYCHOBIOLOGICAL INHIBITION MODEL
THE NEUROBIOLOGICAL MODEL
FROM A CLINICAL POINT OF VIEW

BREAK

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PRIMARY INSOMNIA / INSOMNIA DISORDER

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