Sleep deprivation and Cardiovascular Diseases: up-date and occupational implications

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Summary

- Sleep deprivation: general aspects
- Physiopathological consequences of sleep deprivation
- Sleep deprivation as a cardiovascular and metabolic risk factor
- Sleep deprivation and occupational health

We sleep less than before…

- In 1900 → 9 hous/day
- In 1980 → 7 hours/day
- In 2000 → 6.5 hours/day

- *Schoenborn & Adams, 2010*

National Institute of Health recommends at least

- 10 hours of sleep/night for children
- 9-10 hours of sleep/night for teenagers
- *7-8 hours of sleep/night for adults*

Sleep deprivation

- Impairment of working abilities
- Increase of car accidents
- Strong association with cardiovascular, cerebrovascular and metabolic disorders
- Increased global morbility and mortality

Sleep Deprivation: WHY?

1. Lifestyle
   - Electronic devices
   - Hard work schedules
   - Shift work
2. Sleep disorders
   - *Sleep disordered breathing (SDB)*
   - Insomnia
   - Neurological sleep disorders (periodic limb movements, restless leg syndrome)

3. Ageing

LATEST POPULAR VIDEOS

It's Time to Pay Attention to Sleep, the New Health Frontier

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Study: Fewer Than 1 in 5 Public School Teachers Are Nonwhite

HEALTH SLEEP

It's Time to Pay Attention to Sleep, the New Health Frontier

Alexandra Sifferlin @acsifferlin | April 9, 2014

Your doctor could soon be prescribing crucial shuteye as treatment for everything from obesity to ADHD to mental health as experts say carving out time for sleep is just as important as diet and exercise

MENU TIME

The effect of self-luminous displays on sleep...

A study from the Lighting Research Centre suggests that the light emitted from displays of tablets and similar devices can cause melatonin suppression, which affects our natural body clock.

By using a self-luminous display for two hours before bed, you can suppress melatonin by 22%, making it harder to get to sleep

What is melatonin?

Melatonin is a hormone produced by the pineal gland at night and under the conditions of darkness. It is known as a 'timing messenger', signaling nighttime information throughout the body

Exposure to light at night, especially short-wavelength light, can slow or even decrease melatonin production, interrupting sleep. Suppression of melatonin resulting in circadian disruption has been implicated in sleep disturbances.

It seems we're addicted to our phones...

95% of 18-29yr olds sleep with their phone right next to their bed

25% of people don't silence their phone before going to bed

10% of people are woken up regularly by calls, text or emails

50% will check their phone immediately if they wake in the night

Circulation

Editorials

Sleep Disturbances

Time to Join the Top 10 Potentially Modifiable Cardiovascular Risk Factors?

Susan Redline, MD, MPH;; JoAnne Foody, MD, MPH

Insomnia and the Risk of Acute Myocardial Infarction: A Population Study

Circulation. 2011;124:2073–2081,

Sleep deprivation:

A new cardiovascular risk factor!

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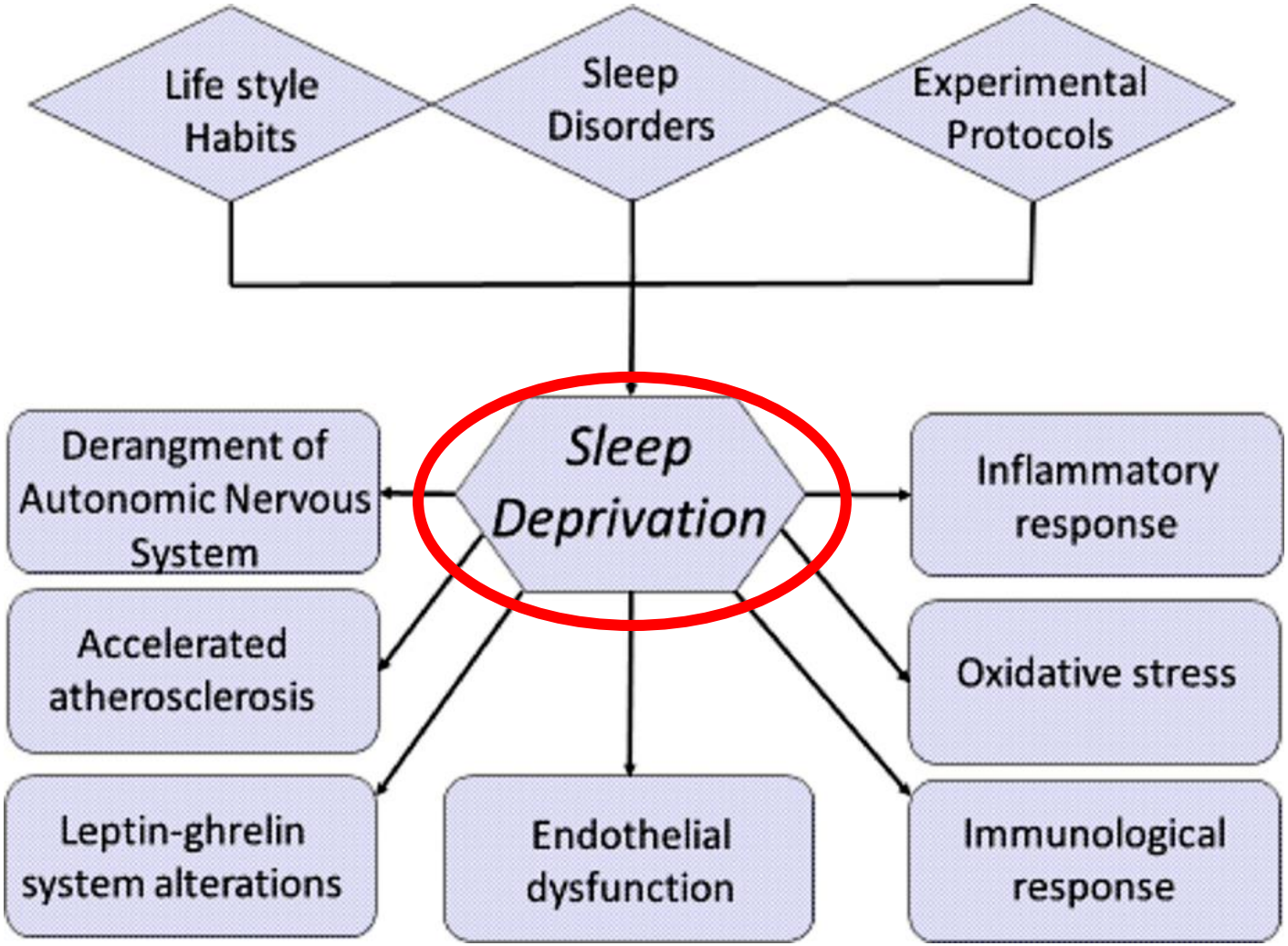

E. Tobaldini et al., Neuroscience and Biobehavioral Reviews, 2017

Increased sympathetic and decreased parasympathetic cardiovascular modulation in normal humans with acute sleep deprivation

		Sleep Deprivation								
		12 h			24 h			36 h		
	Baseline	Δ	CI	*P*	Δ	CI	*P*	Δ	CI	*P*
HR, beats/min	64.9±2.5	−0.82	−3.79, +2.16	0.59	+3.11*	+0.13, +6.09	0.04	−2.21	−5.19, +0.77	0.15
RR, beats/min	17.2±2.9	−1.17*	−1.78, −0.55	<0.001	−0.56	−1.17, −0.06	0.08	−1.55*	−2.17, −0.94	<0.001
SBP, mmHg	119.5±14.0	−0.02	−6.80, +6.76	0.99	+5.08	−1.70,+11.85	0.14	−1.35	−8.13, +5.43	0.69
DBP, mmHg	63.2±11.5	+2.13	−2.99, +7.26	0.41	−0.31	−5.43, +4.82	0.91	+0.49	−4.53, +5.62	0.85
HRV LF(n)	59.39±15.65	+7.73*	+0.40,+15.06	0.039	+7.54*	+0.22,+14.88	0.044	+6.61	−0.72,+13.94	0.077
HRV HF(n)	29.03±11.57	−6.31*	−12.01, −0.62	0.030	−5.35	−11.05, +0.34	0.066	−5.57	−11.26, +0.12	0.055
LF_{R-R}/HF_{R-R}	2.66±1.74	+0.40*	+0.03, +0.76	0.030	+0.33	0.03, +0.70	0.07	+0.36*	0.00, +0.72	0.05
BPV LF (n)	59.41±15.84	+16.02*	+7.49,+24.56	<0.001	+15.43*	+6.89,+23.96	<0.001	+16.55*	+8.01,+25.09	<0.001
BRS	20.1±8.8	−0.18	−0.42, +0.05	0.129	−0.28*	−0.51, −0.04	0.020	−0.18	−0.42, +0.05	0.13

Acute sleep deprivation (36 hours) is associated with increased sympathetic and reduced vagal modulation in healthy subjects

Zhong et al., JAP, 2005

Contents lists available at SciVerse ScienceDirect

European Journal of Internal Medicine

journal homepage: www.elsevier.com/locate/ejim

Original article

One night on-call: Sleep deprivation affects cardiac autonomic control and inflammation in physicians☆

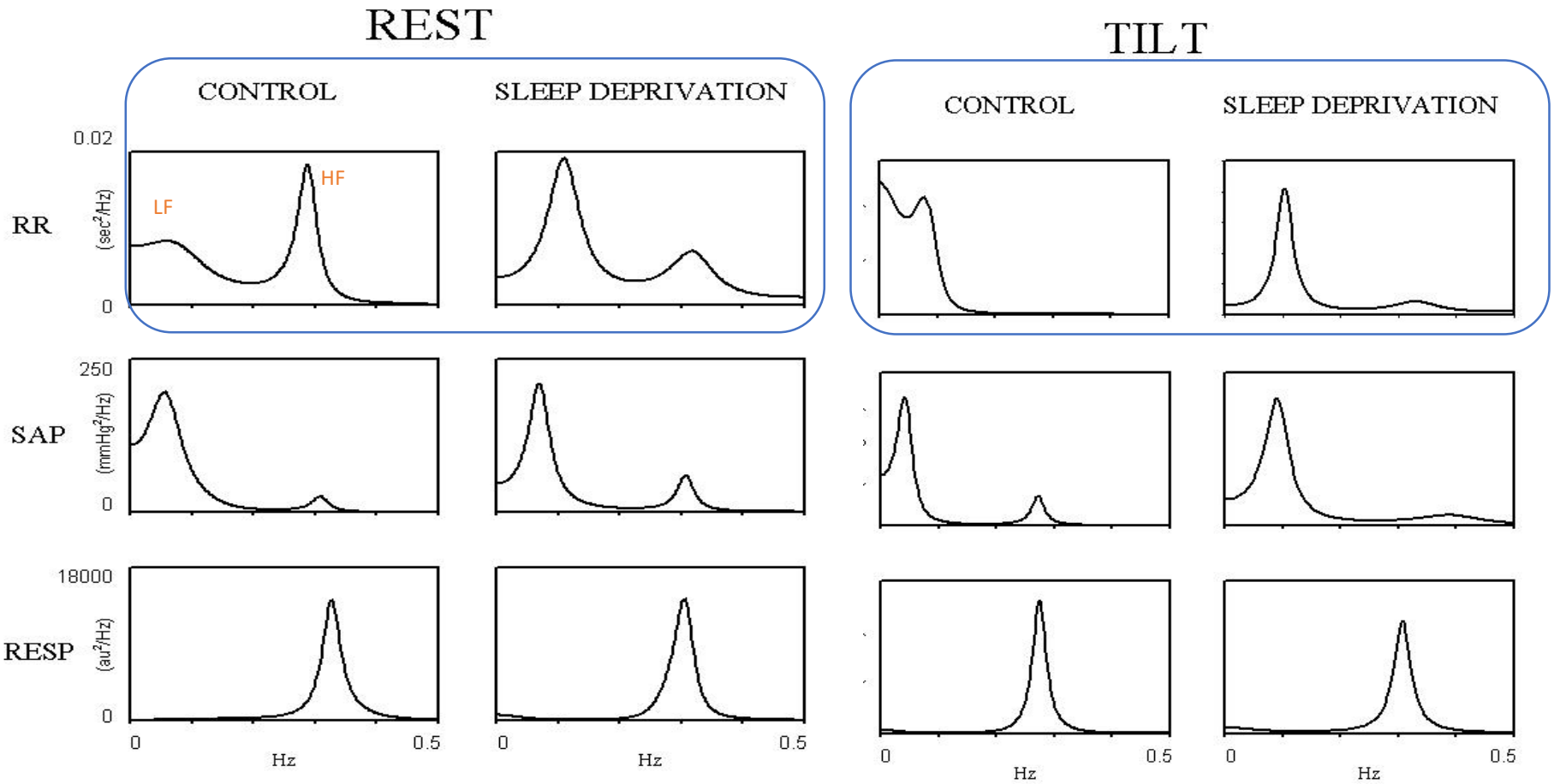

Table 2. *Change in plasma levels of biological parameters at 0800*

	Before TSD (D1)	After TSD (D3)	Recovery (D4)	Friedman *P* Value
ICAM-1, ng/ml	215 ± 13	227 ± 15	253 ± 16*	0.02
VCAM, ng/ml	491 ± 27	489 ± 24	513 ± 34	NS
E-selectin, ng/ml	35 ± 6	54 ± 8*	42 ± 6	0.02
CRP, μg/ml	1.22 ± 0.46	0.55 ± 0.13	0.61 ± 0.14	NS
IL-6, pg/ml	0.60 ± 0.13	0.62 ± 0.10	1.20 ± 0.23*	0.03
TNF-α, pg/ml	0.88 ± 0.32	1.05 ± 0.30	0.88 ± 0.24	NS
Cortisol, mmol/l	524 ± 42	491 ± 27	503 ± 16	NS
Epinephrine, pg/ml	81 ± 16	68 ± 10	57 ± 13	NS
Norepinephrine, pg/ml	757 ± 110	885 ± 121	1,113 ± 125*	0.04

Sleep deprivation and inflammation

Journal of the American College of Cardiology
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Vol. 43, No. 4, 2004
ISSN 0735-1097/04/$30.00
doi:10.1016/j.jacc.2003.07.050

Sleep Loss and Inflammatory Markers

Effect of Sleep Loss on C-Reactive Protein, an Inflammatory Marker of Cardiovascular Risk

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Meredith M. Regan, SCD,§ Nick J. Price,‖ David F. Dinges, PHD,¶ Janet M. Mullington, PHD#
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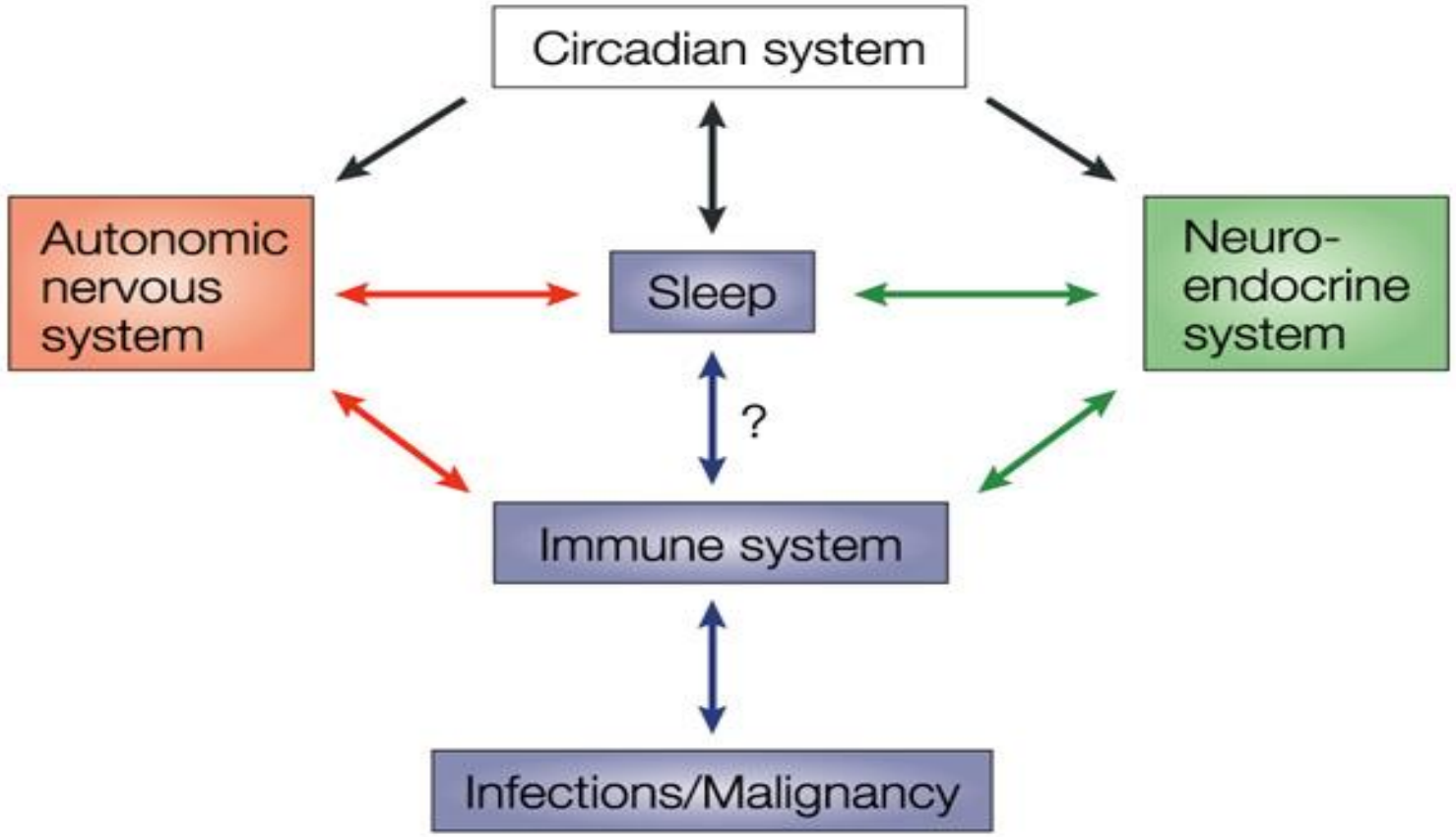
7

Sleep loss and inflammation

- Increased production of pro-inflammatory cytokines (IL-1, IL-6, TNF-α)
- Deregulation of natural killer cells
- Neutrophilic degranulation

"Sick and tired"



Nature Reviews | Immunology

Summary

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Sleep Duration and All-Cause Mortality: A Systematic Review and Meta-Analysis of Prospective Studies

Francesco P. Cappuccio, MD, FRCP[1]; Lanfranco D'Elia, MD[2]; Pasquale Strazzullo, MD[2]; Michelle A. Miller, PhD[1]

[1]University of Warwick, Warwick Medical School, Clinical Sciences Research Institute, Coventry, UK; [2]Department of Clinical and Experimental Medicine, "Federico II" University of Naples Medical School, Naples, Italy

Background: Increasing evidence suggests an association between both short and long duration of habitual sleep with adverse health outcomes.

Objectives: To assess whether the population longitudinal evidence supports the presence of a relationship between duration of sleep and all-cause mortality, to investigate both short and long sleep duration and to obtain an estimate of the risk.

Methods: We performed a systematic search of publications using MEDLINE (1966-2009), EMBASE (from 1980), the Cochrane Library, and manual searches without language restrictions. We included studies if they were prospective, had follow-up >3 years, had duration of sleep at baseline, and all-cause mortality prospectively. We extracted relative risks (RR) and 95% confidence intervals (CI) and pooled them using a random effect model. We carried out sensitivity analyses and assessed heterogeneity and publication bias.

Results: Overall, the 16 studies analyzed provided 27 independent cohort samples. They included 1,382,999 male and female participants (follow-up range 4 to 25 years), and 112,566 deaths. Sleep duration was assessed by questionnaire and outcome through death certification. In the pooled analysis, short duration of sleep was associated with a greater risk of death (RR: 1.12; 95% CI 1.06 to 1.18; P < 0.01) with no evidence of publication bias (P = 0.74) but heterogeneity between studies (P = 0.02). Long duration of sleep was also associated with a greater risk of death (1.30; [1.22 to 1.38]; P < 0.0001) with no evidence of publication bias (P = 0.18) but significant heterogeneity between studies (P < 0.0001).

Conclusion: Both short and long duration of sleep are significant predictors of death in prospective population studies.

Keywords: Sleep duration, mortality, meta-analysis

Sleep Duration and Cardiovascular Disease: Results from the National Health Interview Survey

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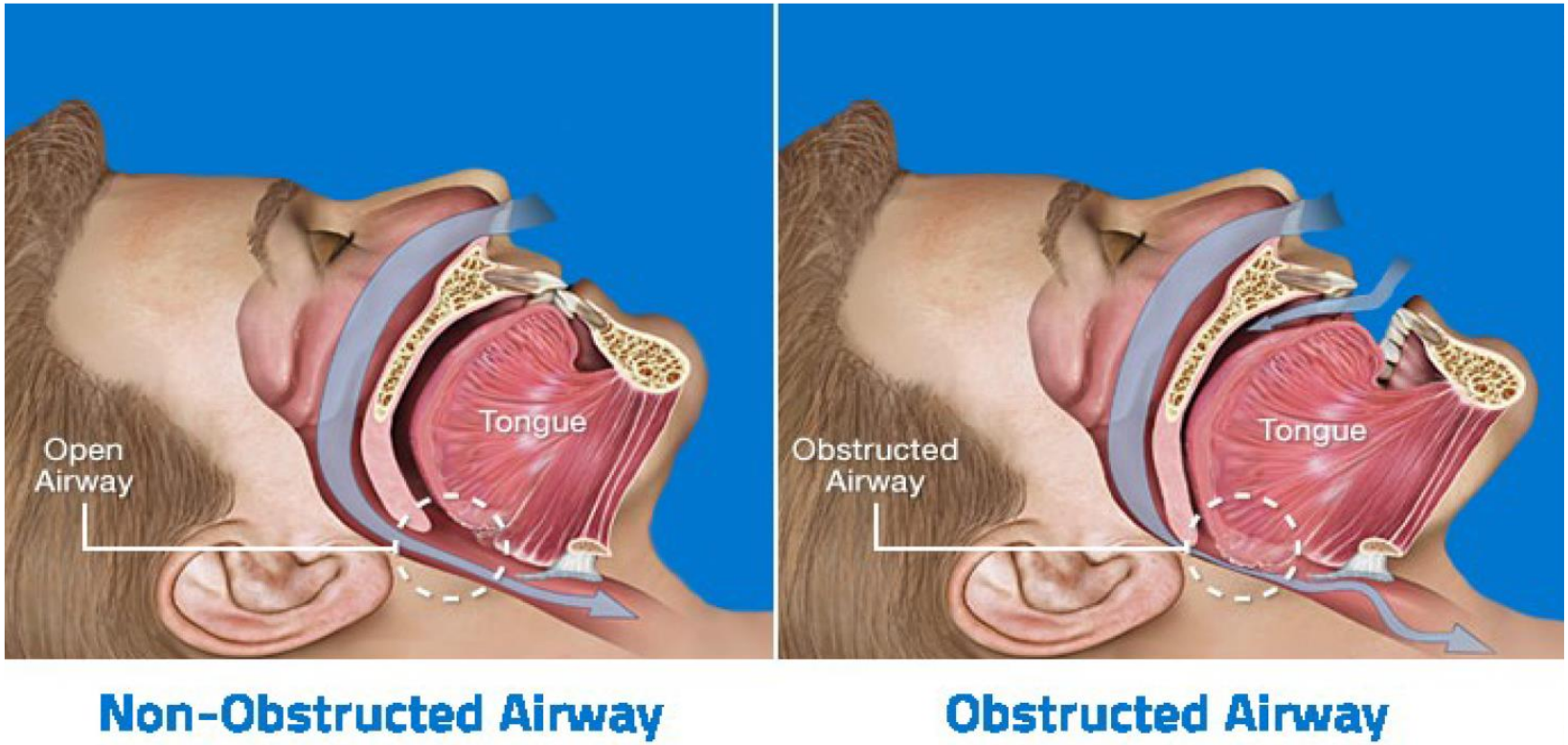
Background: Previous studies have shown that both short and long sleep durations are related to increased likelihood of diabetes and hypertension. However, the relation between sleep duration and cardiovascular disease (CVD) is not clear. We examined the hypothesis that compared with sleep duration of 7 hours, shorter and longer sleep durations are independently related to CVD.

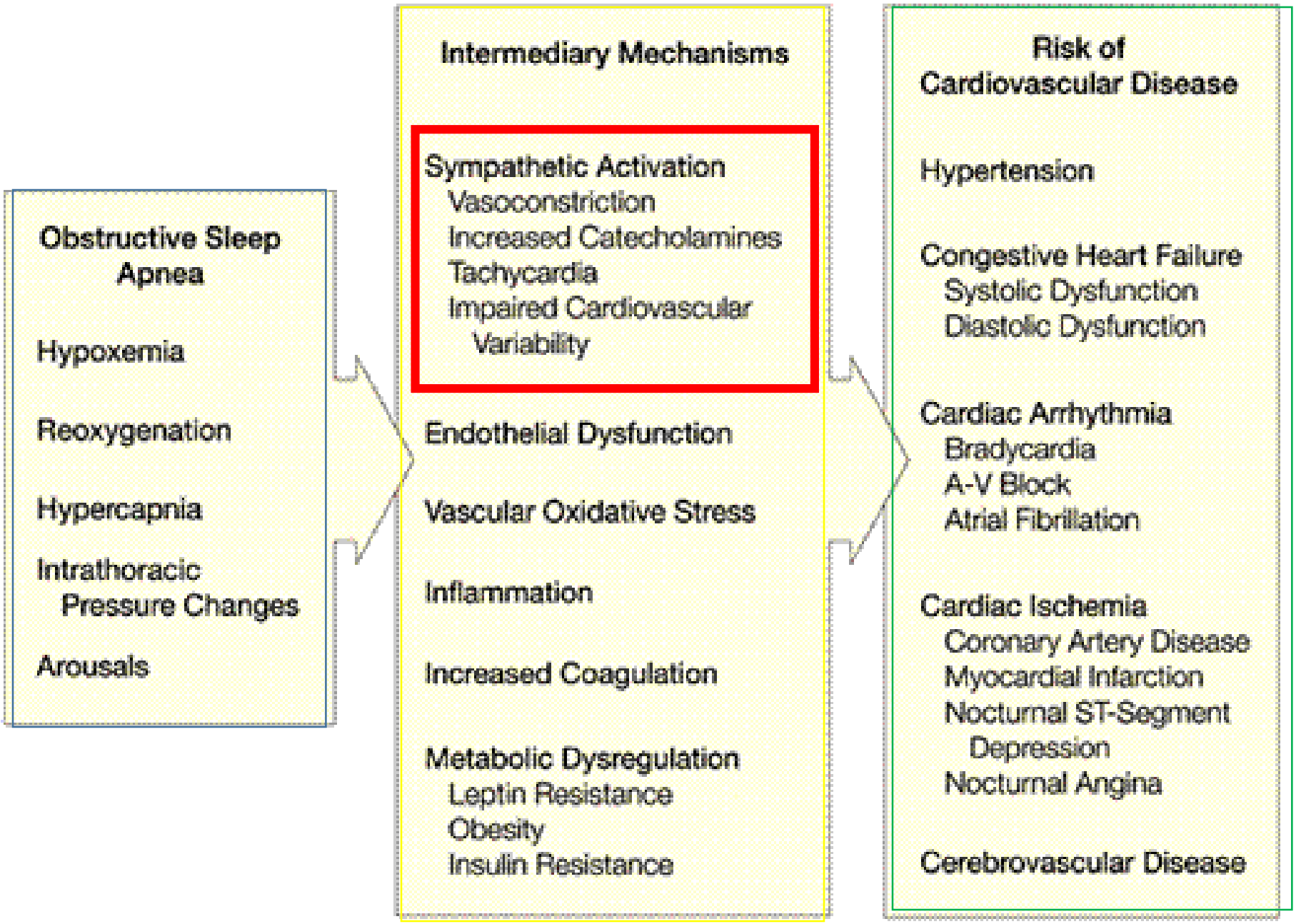
Methods: We conducted a cross-sectional study of 30,397 National Health Interview Survey 2005 participants ≥ 18 years of age (57.1% women). Sleep duration was categorized as ≤ 5 hours, 6 hours, 7 hours, 8 hours, and ≥ 9 hours. The main outcome of interest was the presence of any CVD (n = 2146), including myocardial infarction, angina, and stroke.

Results: We found both short and long sleep durations to be independently associated with CVD, independent of age, sex, race-ethnicity, smoking, alcohol intake, body mass index, physical activity, diabetes mellitus, hypertension, and depression. Compared with a sleep duration of 7 h (referent), the multivariate odds ratio (95% confidence interval) of CVD was 2.20 (1.78, 2.71), 1.33 (1.13, 1.57), 1.23 (1.06, 1.41), and 1.57 (1.31, 1.89) for sleep duration ≤ 5 h, 6 h, 8 h, and ≥ 9 h. This association persisted in subgroup analyses by gender, race-ethnicity, and body mass index categories. Also, similar associations were observed when we examined myocardial infarction and stroke separately.

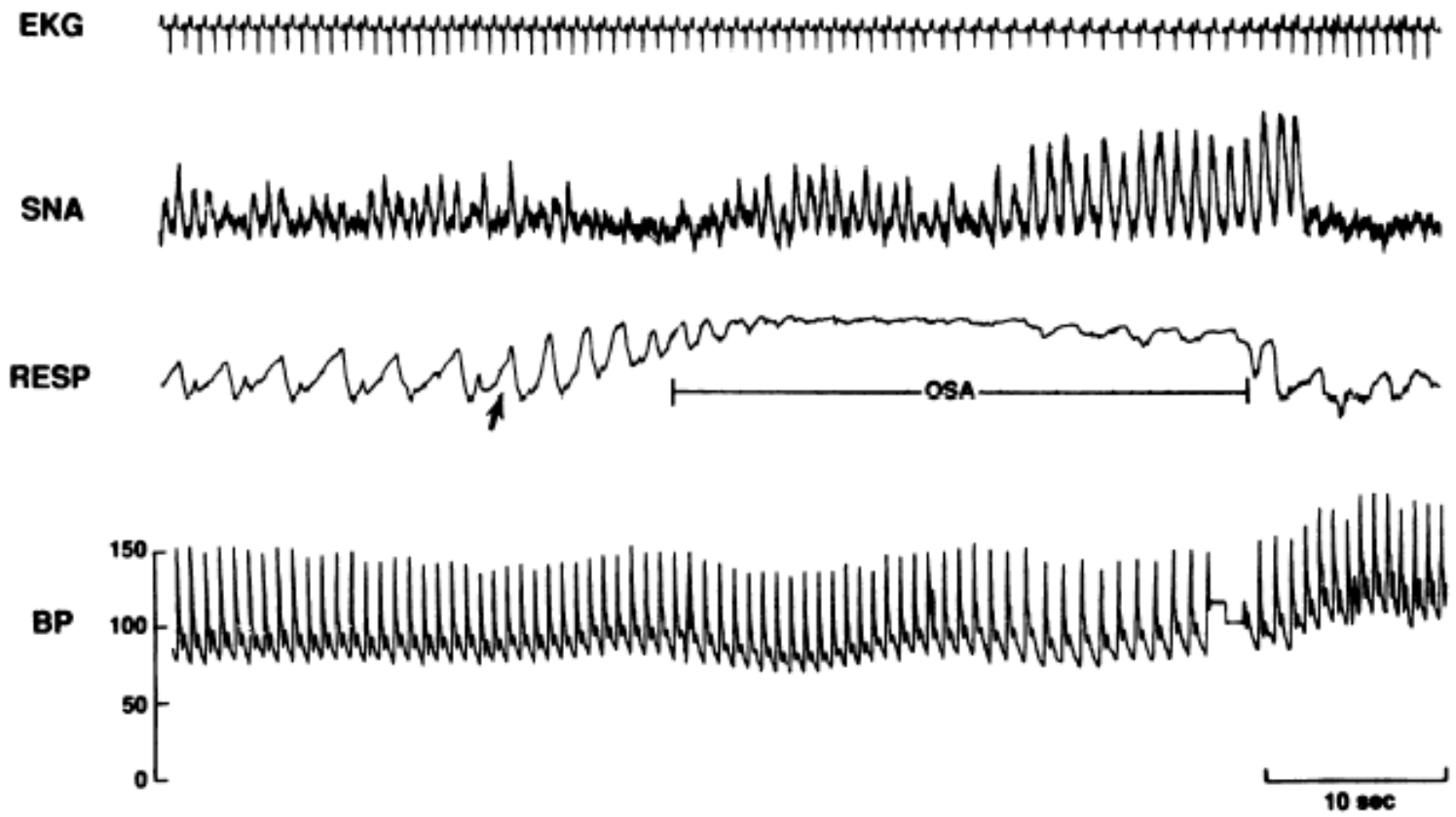
Conclusion: Compared with sleep duration of 7 h, there was a positive association between both shorter and longer sleep durations and CVD in a representative sample of US adults. These results suggest that sleep duration may be an important marker of CVD.

Obstructive Sleep-Apnea



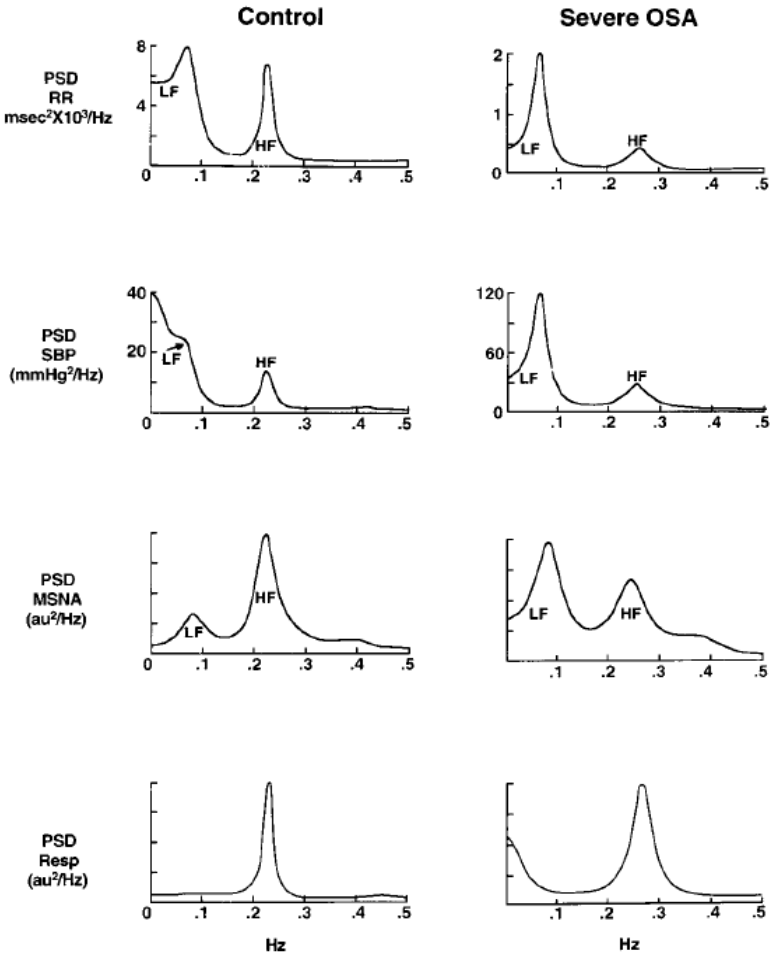

Obstructive Sleep Apnea
Hypoxemia
Reoxygenation
Hypercapnia
Intrathoracic Pressure Changes
Arousals
Intermediary Mechanisms
Sympathetic Activation
Vasoconstriction
Increased Catecholamines
Tachycardia
Impaired Cardiovascular Variability
Endothelial Dysfunction
Vascular Oxidative Stress
Inflammation
Increased Coagulation
Metabolic Dysregulation
Leptin Resistance
Obesity
Insulin Resistance
Risk of Cardiovascular Disease
Hypertension
Congestive Heart Failure
Systolic Dysfunction
Diastolic Dysfunction
Cardiac Arrhythmia
Bradycardia
A-V Block
Atrial Fibrillation
Cardiac Ischemia
Coronary Artery Disease
Myocardial Infarction
Nocturnal ST-Segment Depression
Nocturnal Angina
Cerebrovascular Disease

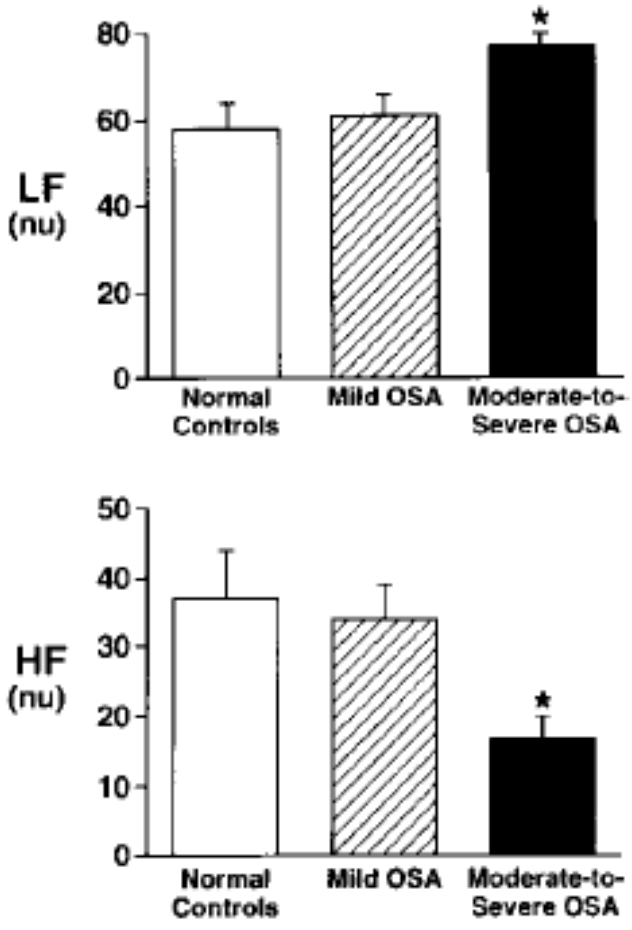
Apneas and Autonomic Nervous System



Altered Cardiovascular Variability in Obstructive Sleep Apnea

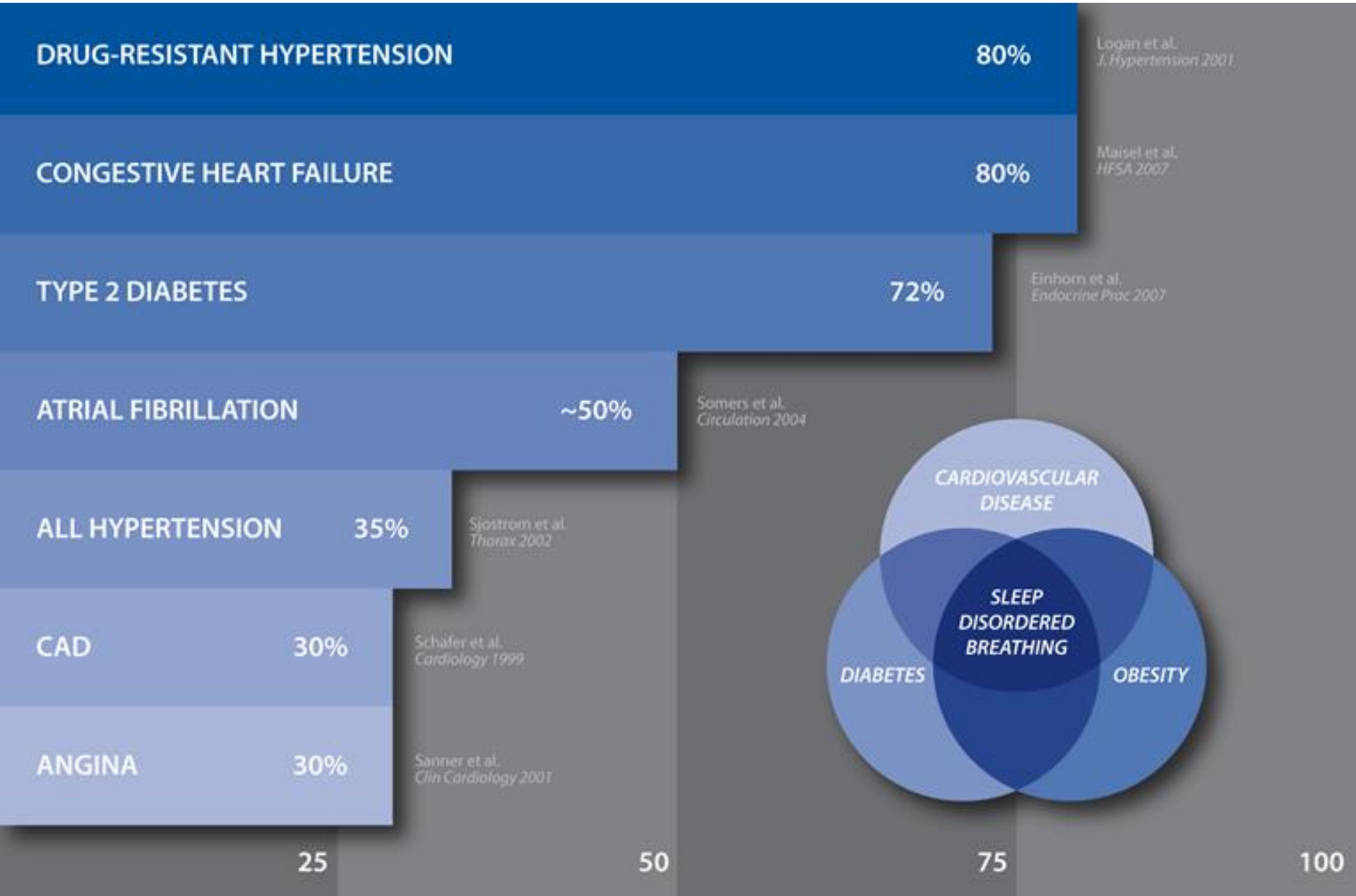
Krzysztof Narkiewicz, MD, PhD; Nicola Montano, MD, PhD; Chiara Cogliati, MD;
Philippe J.H. van de Borne, MD, PhD; Mark E. Dyken, MD; Virend K. Somers, MD, PhD





(*Circulation*. 1998;98:1071-1077.)

OSA and cardiovascular disorders

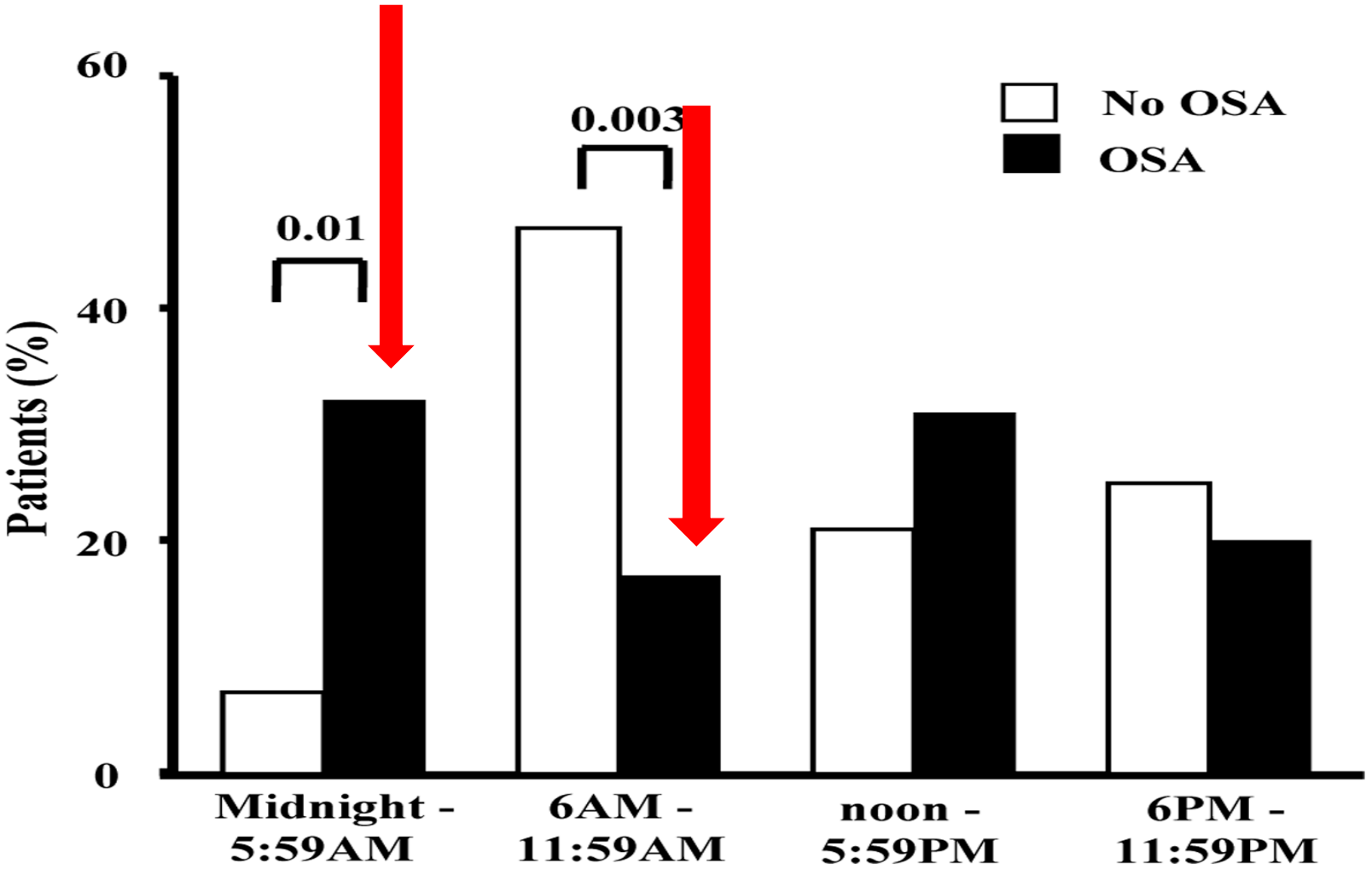


“OSA as the first identifiable cause of hypertension”

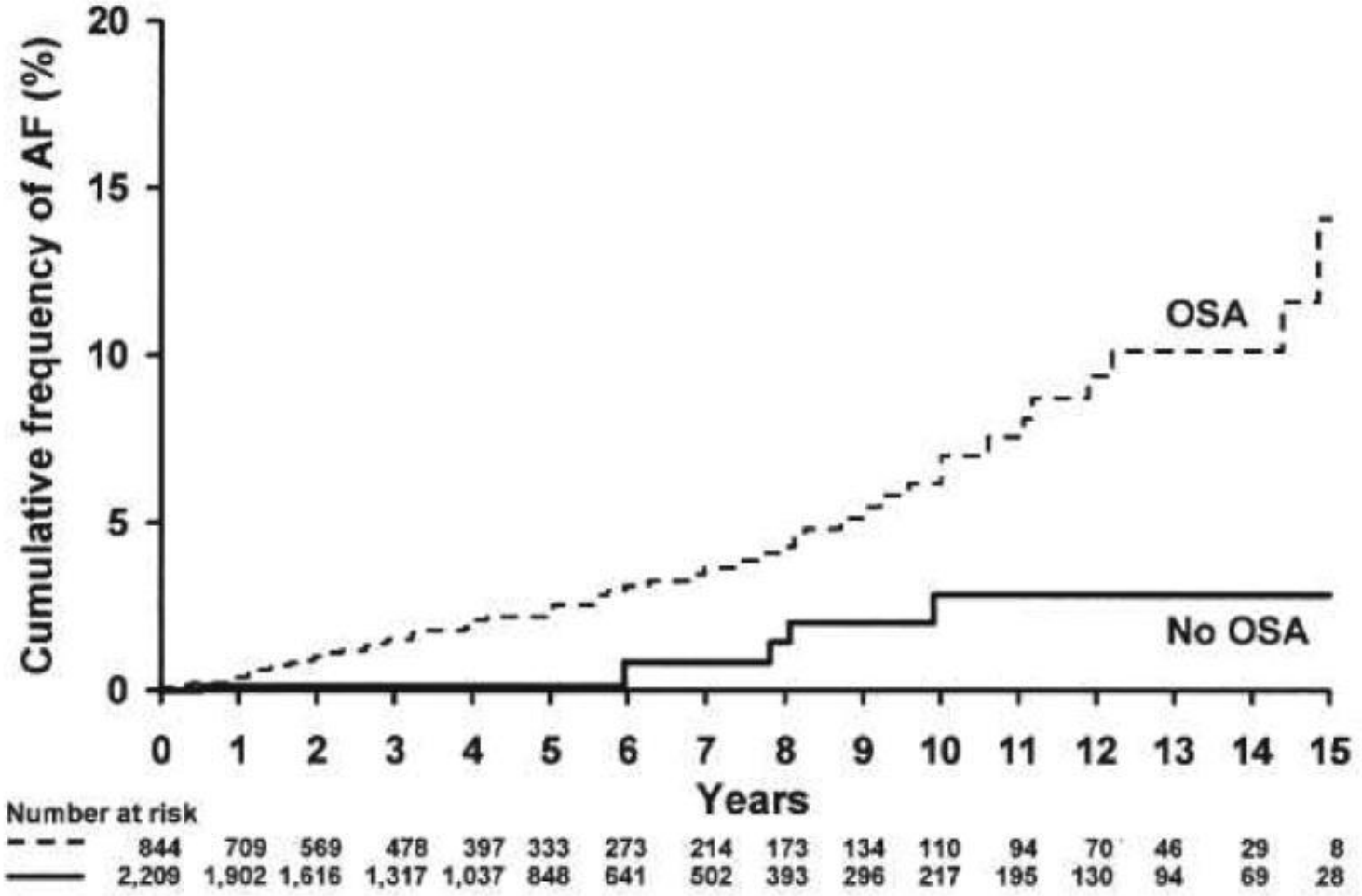
Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of Hight Blood Pressure

(Chobanian AV. *JAMA* 2003, 289:2560)

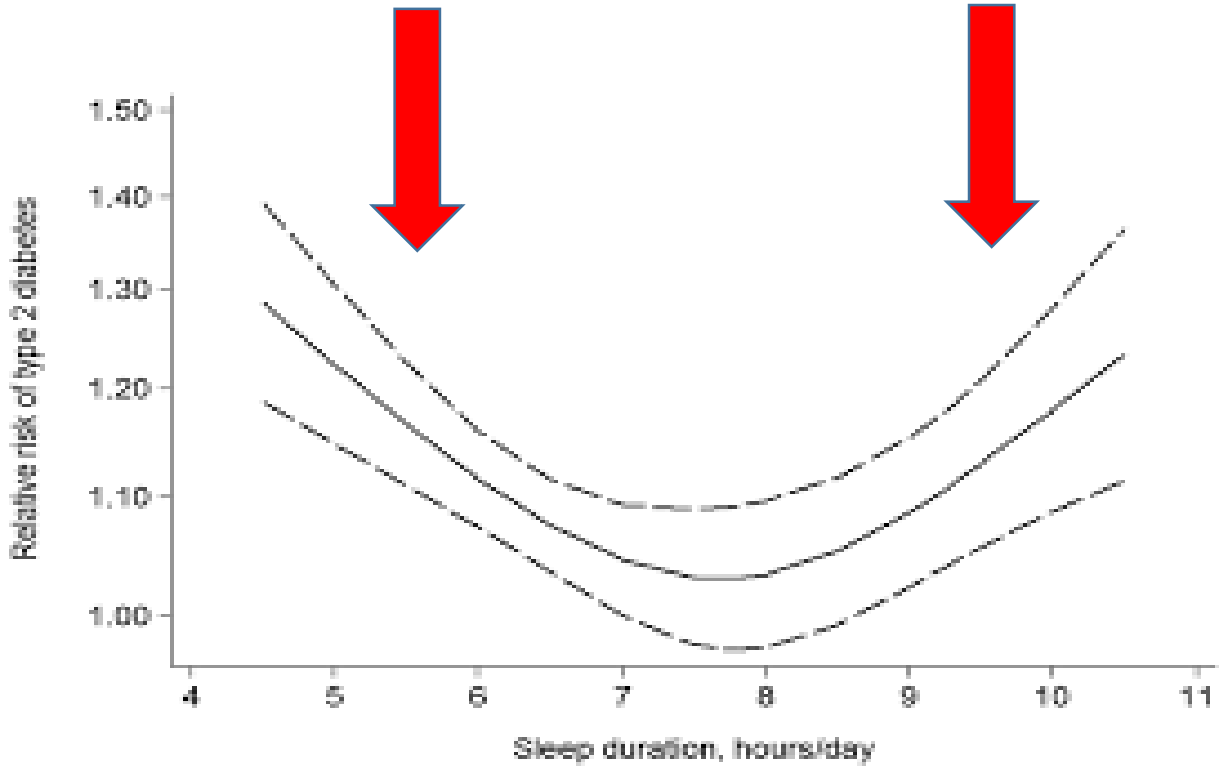
OSA and myocardial infarction



OSA and atrial fibrillation

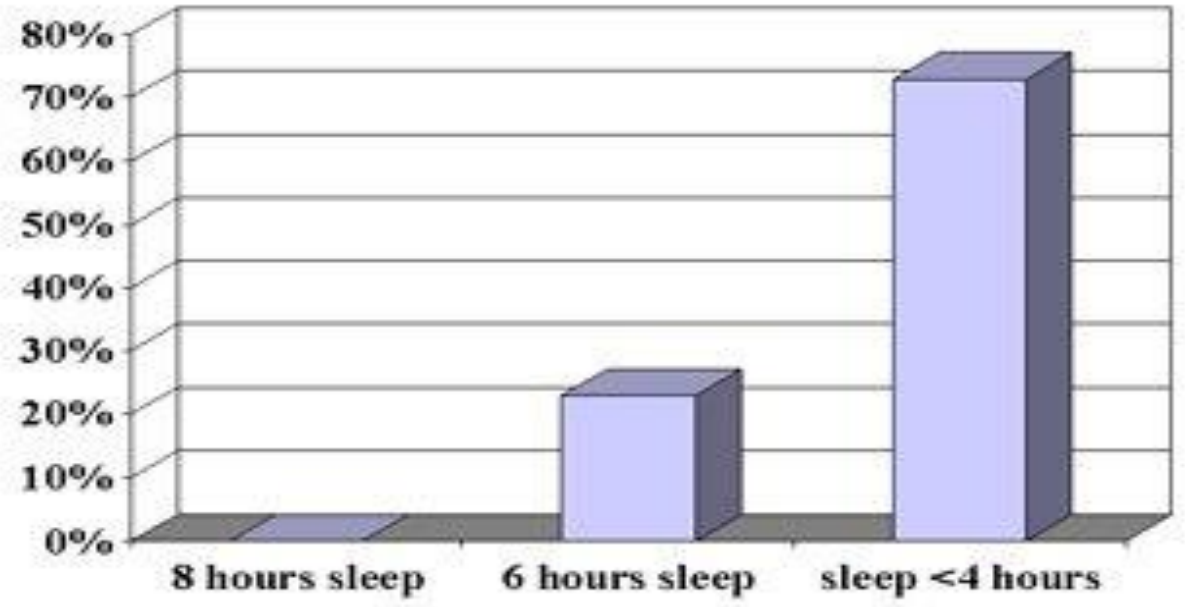


Sleep duration and diabetes

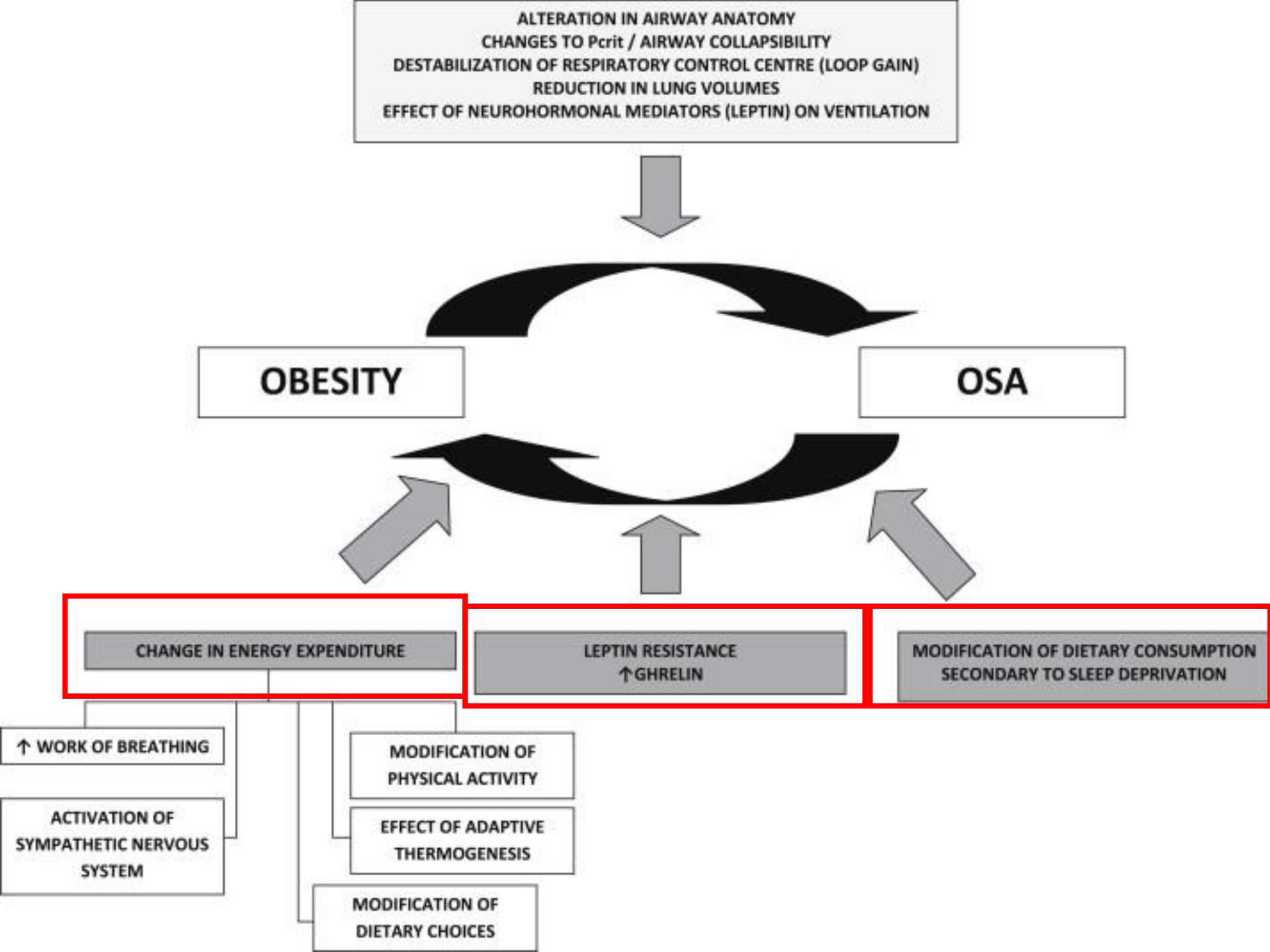


(Anothaisintawee, Sleep Medicine Reviews 2016)

Sleep duration and obesity



(Wu et al., Sleep Medicine 2014)


ALTERATION IN AIRWAY ANATOMY
CHANGES TO Pcrit / AIRWAY COLLAPSIBILITY
DESTABILIZATION OF RESPIRATORY CONTROL CENTRE (LOOP GAIN)
REDUCTION IN LUNG VOLUMES
EFFECT OF NEUROHORMONAL MEDIATORS (LEPTIN) ON VENTILATION
OBESITY
OSA
CHANGE IN ENERGY EXPENDITURE
LEPTIN RESISTANCE
↑GHRELIN
MODIFICATION OF DIETARY CONSUMPTION SECONDARY TO SLEEP DEPRIVATION
↑ WORK OF BREATHING
ACTIVATION OF SYMPATHETIC NERVOUS SYSTEM
MODIFICATION OF PHYSICAL ACTIVITY
EFFECT OF ADAPTIVE THERMOGENESIS
MODIFICATION OF DIETARY CHOICES

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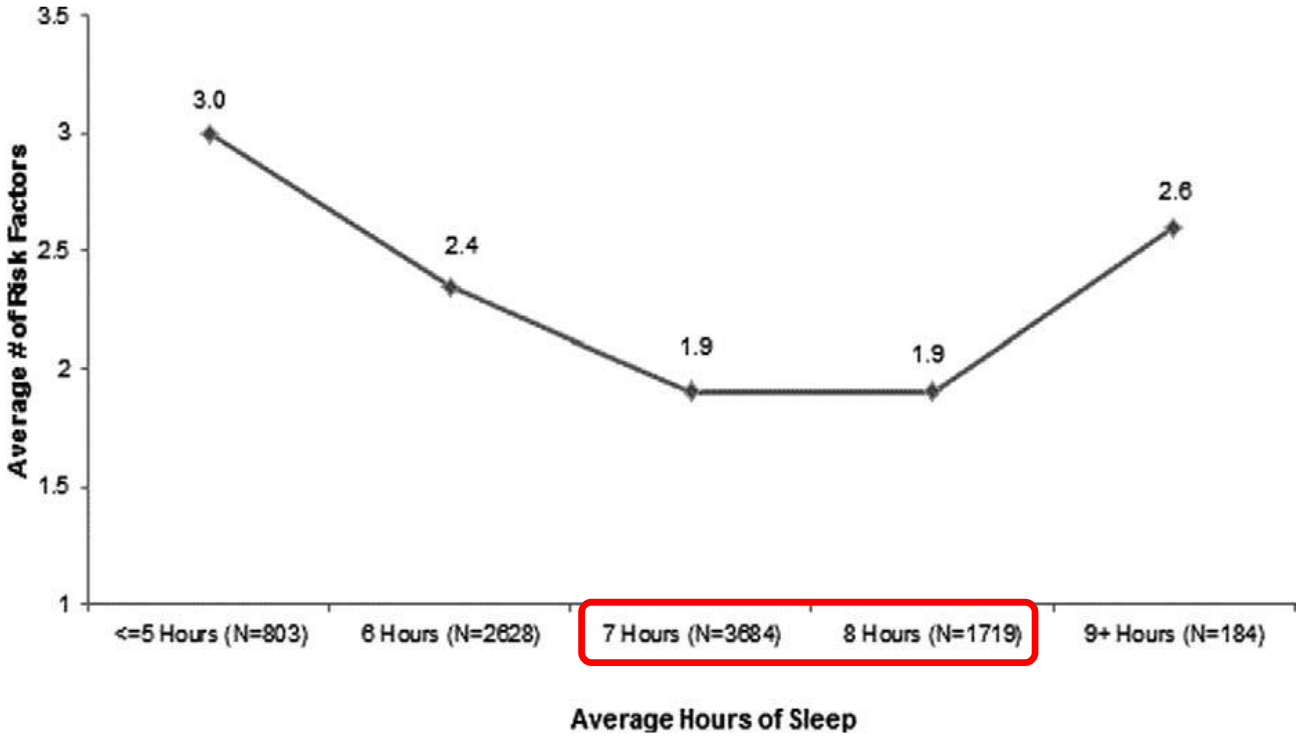
- adverse health outcomes
- cardiovascular disease
- obesity, diabetes, depression, and anxiety
- safety issues related to drowsy driving and injuries

- Analysis of 2013 and 2014 Behavioral Risk Factor Surveillance System data conducted to examine 93 detailed occupation groups in 29 states
- Prevalence of <7 hours of sleep per day (short sleep duration): from 21.4% among air transportation workers to 58.2% among communications equipment workers.
- Highest prevalence of short sleep duration were among workers in the following five groups: Production (42.9%), Healthcare Support (40.1%), Healthcare Practitioners and Technical (40.0%), Food Preparation and Serving-Related (39.8%), and Protective Service (39.2%)
- Workers in occupations with high prevalence of short sleep duration might be most at risk for sleep-related accidents and adverse health outcomes associated with short sleep duration.
- Work-related factors should be further evaluated in the context of short sleep duration to guide prevention efforts.

Shockey et al., 2017

Relationship between health care costs, short-term disability, absenteeism, and presenteeism (on-the-job work loss) and the hours of sleep



Worksite wellness programs often address health risks and medical conditions and ***may benefit from incorporating sleep education***

Sleep Breath (2015) 19:35–44
DOI 10.1007/s11325-014-1015-8

REVIEW

Occupational health of patients with obstructive sleep apnea syndrome: a systematic review

Ottavia Guglielmi · Bernabé Jurado-Gámez · Francisco Gude · Gualberto Buela-Casal

- Effects on working skills: reduction of working abilities and perfomances (concentation, learning, organization)
- Reduction of productivity → more working days lost
- CPAP improves working perfomances, reduces hypersomnolence, mood disturbances and has positive effects on cardiovascular and metabolic risks