

Epidémiologie des troubles du sommeil en Europe

Pr Raphael Heinzer

Centre d'investigation et de recherche sur le sommeil (CIRS)

CHUV, Lausanne, Suisse

Séminaire sommeil France-Afrique

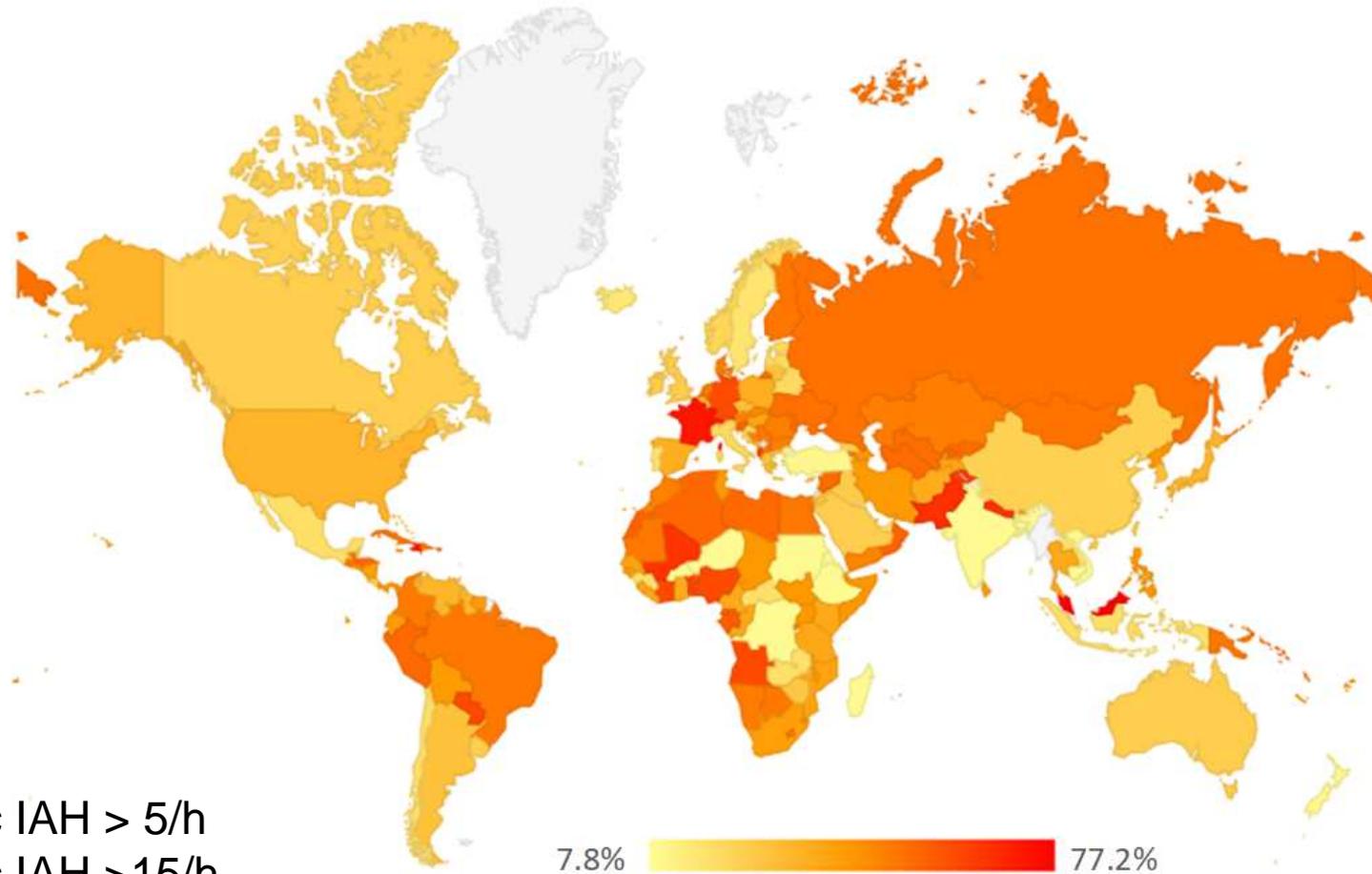
Cotonou 2.05.2024

Que nous apprennent les cohortes européennes en termes de:

- 1) Prévalence des troubles respiratoires nocturnes (SAOS) ?
- 2) Valeur de l'IAH dans l'évaluation de la sévérité du SAOS et de ses conséquences ?
- 3) Facteurs prédictifs du risque cardiovasculaire associé au SAOS ?

Prévalence du SAOS en population générale

Estimation du nombre de personnes atteintes de SAS au niveau mondial



936 millions avec IAH > 5/h
424 millions avec IAH > 15/h

Adam V Benjafield. Lancet resp med 2019

Etude HypnoLaus

-

-

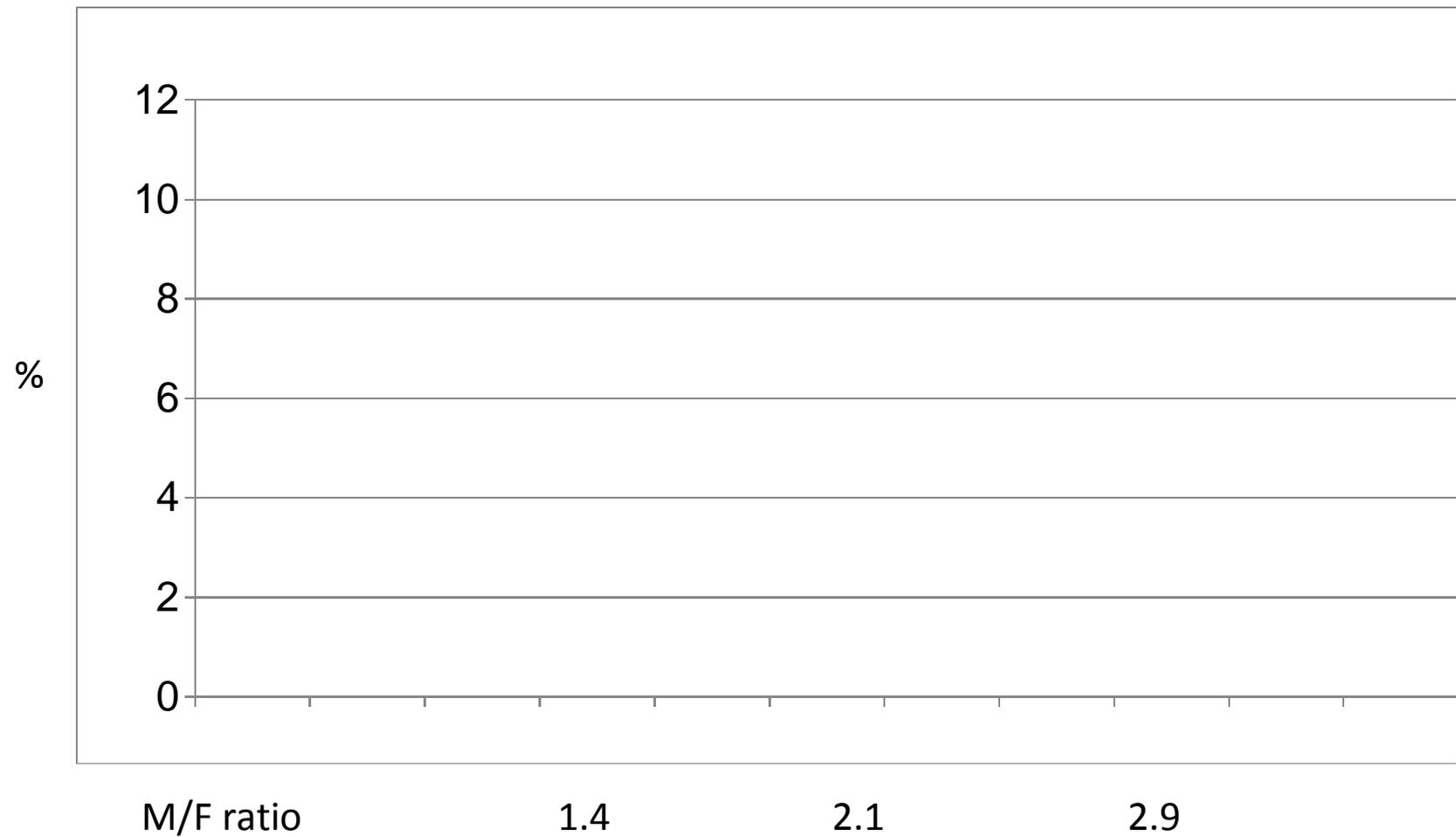


Etude HypnoLaus

Parameters	Value
Nombre de participants	2162
Age (y)	58.5 ± 11.0
Sexe	50.4 % femmes
IMC (kg/m ²)	26.2 ± 4.4
Score d'Epworth	6.2 ± 3.9
Tour de cou (cm)	36.9 ± 3.9

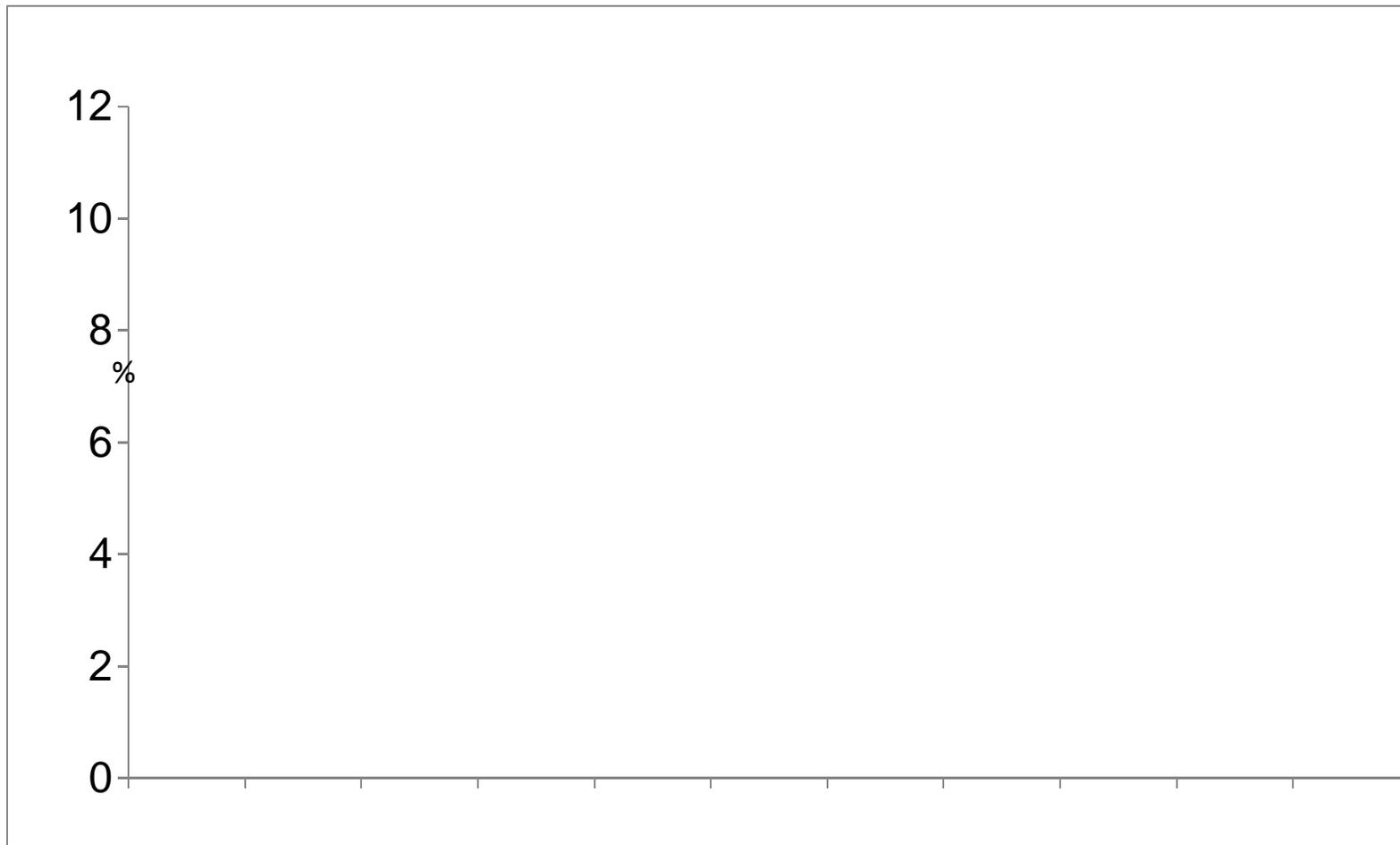
Prevalence des apnées du sommeil à Lausanne

HypnoLaus Cohort (N = 2121) age: 40-85 ans



Prévalence d'apnées du sommeil + somnolence (Epworth >10/24)

HypnoLaus Cohort (N = 2121) age: 40-85 ans



Prevalence and association analysis of obstructive sleep apnea with gender and age differences – Results of SHIP-Trend

Ingo Fietze¹ | Naima Laharnar¹  | Anne Obst² | Ralf Ewert² | Stephan B. Felix² | Carmen Garcia¹ | Sven Gläser^{2,3} | Martin Glos¹ | Carsten Oliver Schmidt⁴ | Beate Stubbe² | Henry Völzke⁴ | Sandra Zimmermann¹ | Thomas Penzel¹

JSR 2018

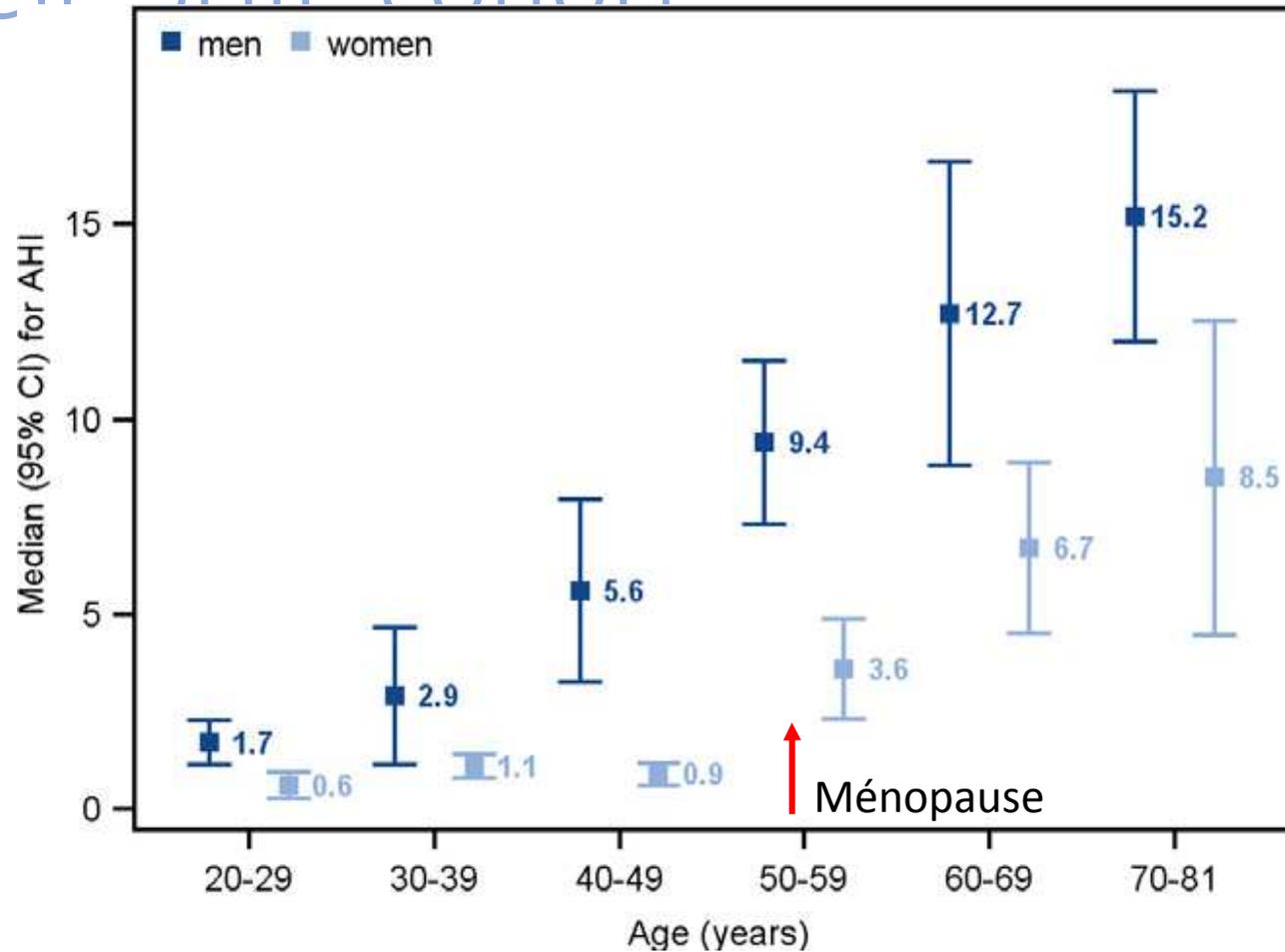
- 1208 participants. 20 - 81 years old (54% men, median age 54 years)
- Overnight polysomnography

AHI \geq 5/h: 46% (59% men, 33% women)

AHI \geq 15/h 21% (30% men, 13% women)

AHI \geq 5 et Epworth $>$ 10 6.3 % (9% men, 3% women)

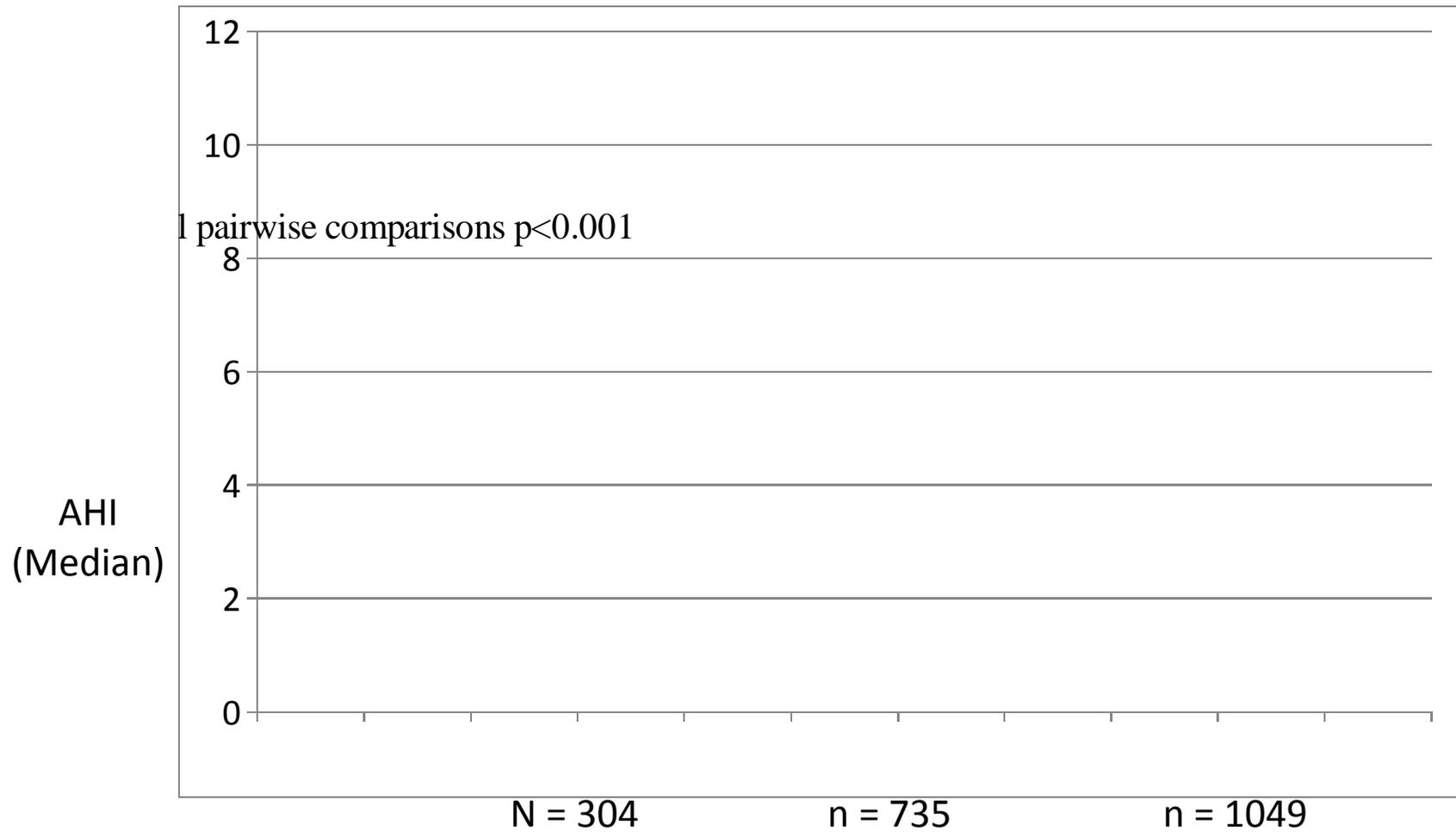
Effet de l'âge sur la prévalence des apnées du sommeil: SHIP cohort



NB: non ajusté pour le BMI etc

Fietze I. JSR 2018

OSA et



Ce qu'il faut retenir

Dans les cohortes populationnelles, on trouve fréquemment un IAH élevé.

- Prévalence d'un IAH >15/h: 9.9% à 23% des femmes
14.5% à 49% des hommes
- Augmente avec l'âge, l'obésité, le sexe masculin et la ménopause
- Beaucoup de patients ne sont pas somnolents et ne présentent pas de plaintes liées au SAOS !

Questions

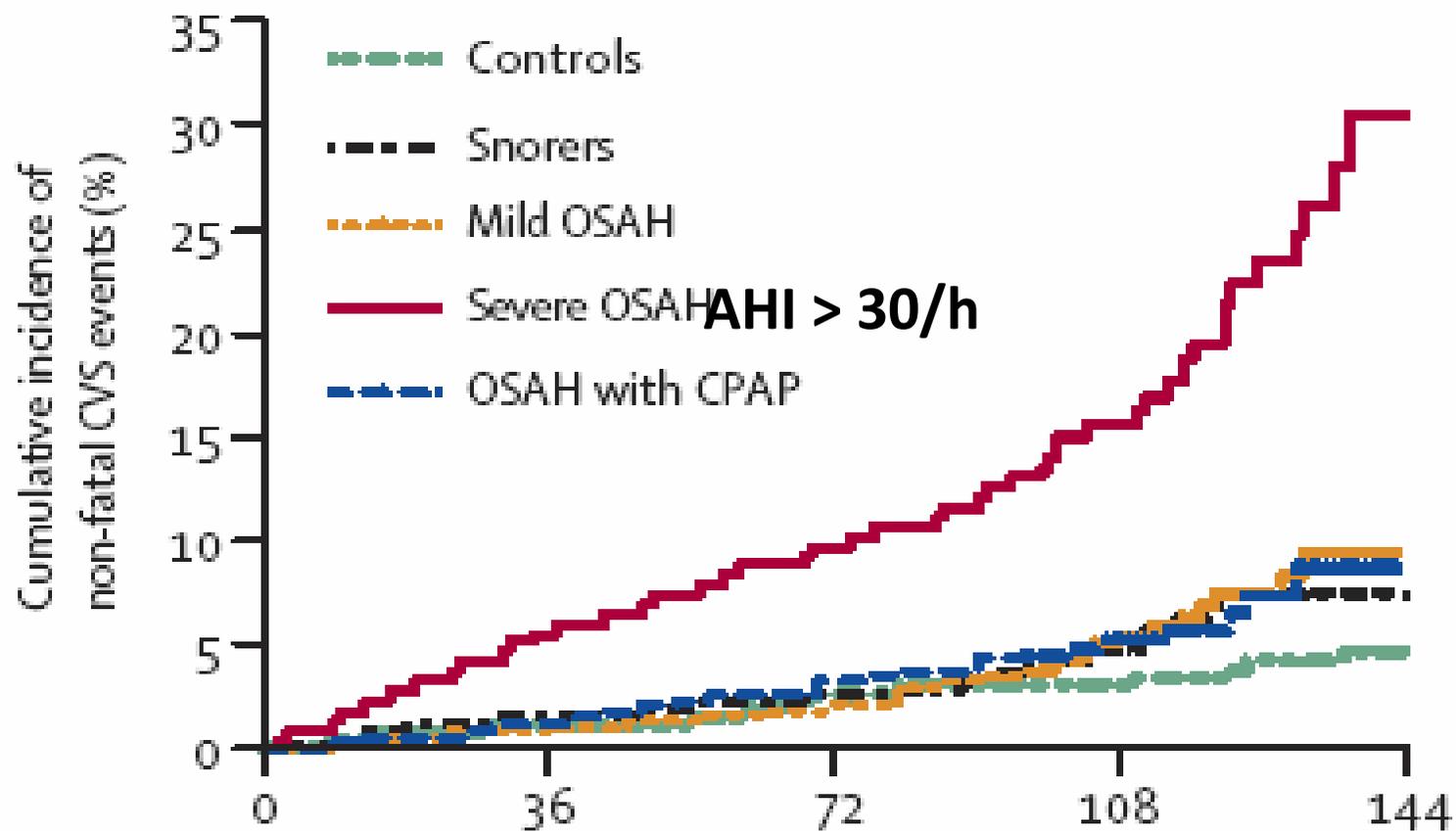
Est-ce que l'IAH est un bon marqueur de la sévérité du SAOS ?

Est-ce que l'IAH permet de prédire le risque cardiovasculaire lié au SAOS ?

Parmi les patients SAOS non-symptomatiques, qui doit être traité ?

Incidence d'événements cardiovasculaires en fonction de l'IAH dans une population masculine

B

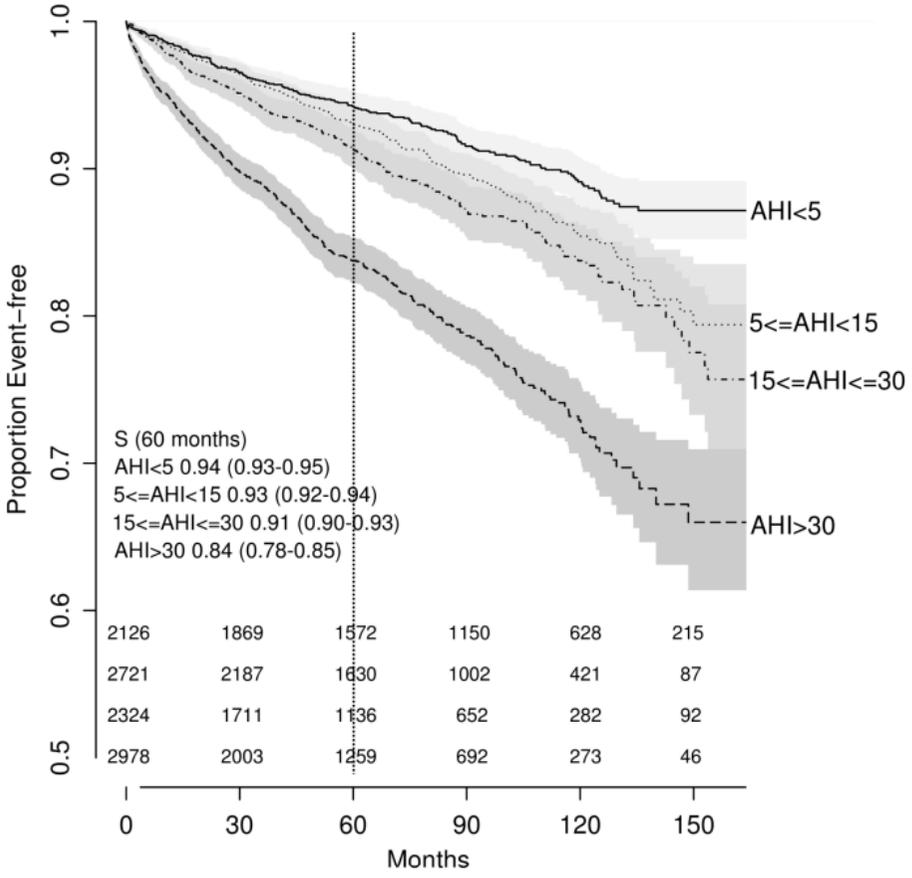


Marin et al. The Lancet 2005

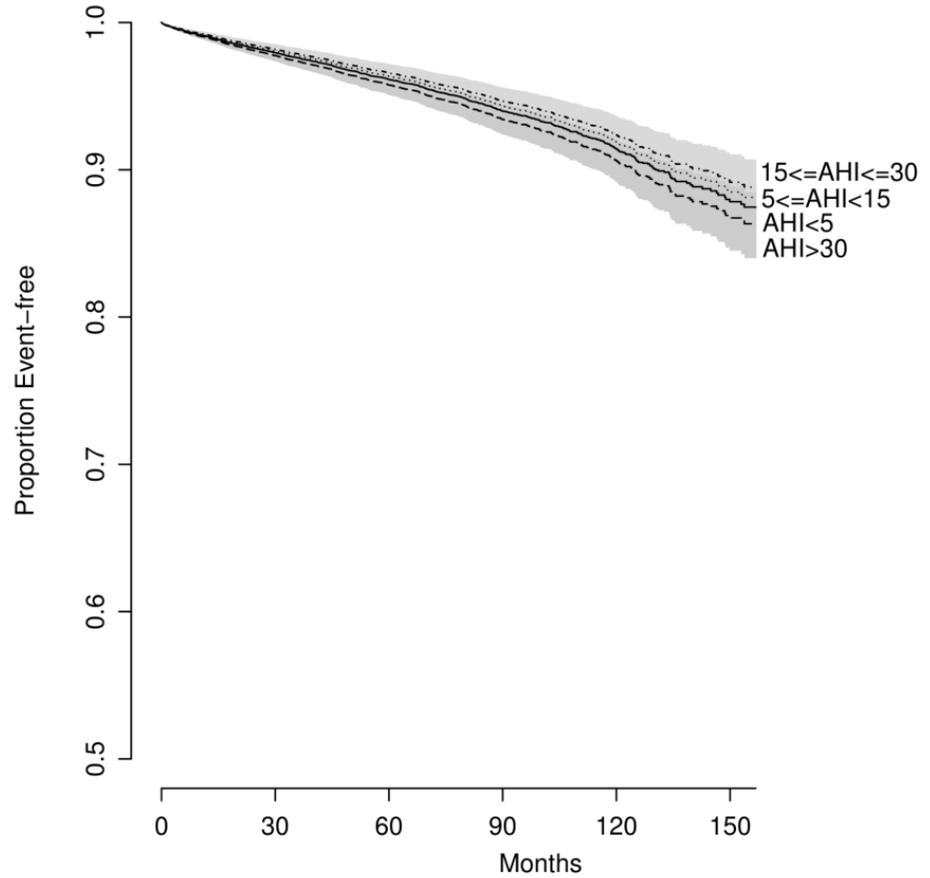
Obstructive Sleep Apnea and Risk of Cardiovascular Events and All-Cause Mortality: A Decade-Long Historical Cohort Study

Kendzerska T. PLoS One 2014

Cohorte historique sur 16 ans : N = 10149. Outcome composite: événements et mortalité CV



Non-ajusté pour les FRCV

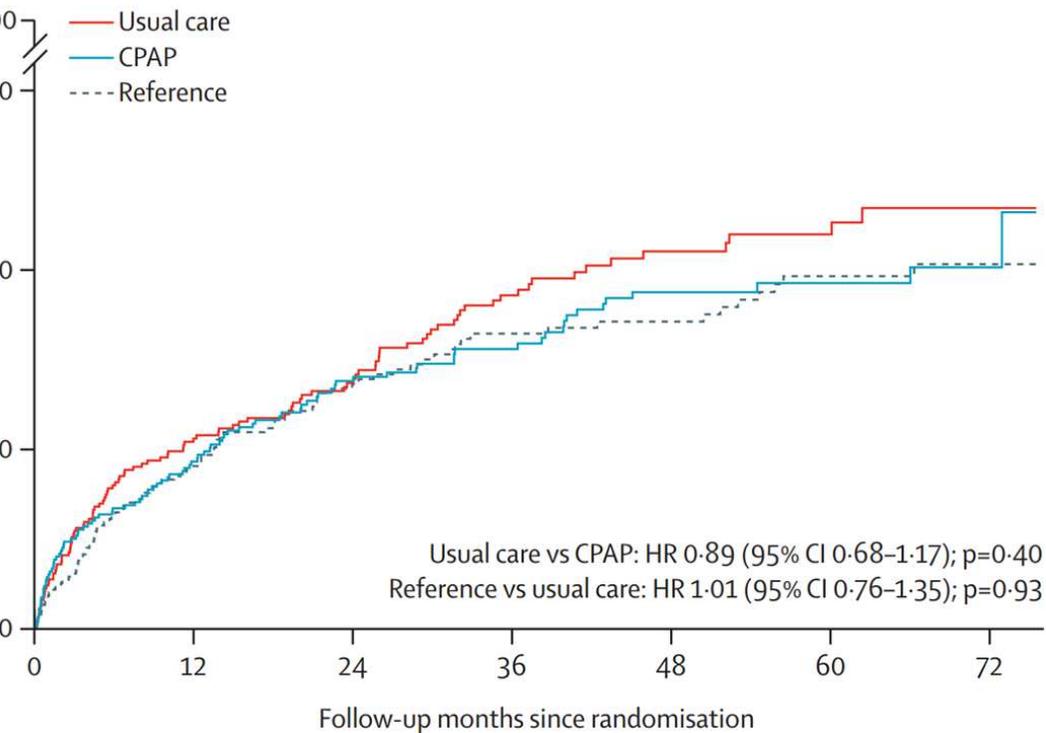


Ajusté pour les FRCV

IAH et incidence des événements cardiovasculaires

ISAACC: population cardiovasculaire

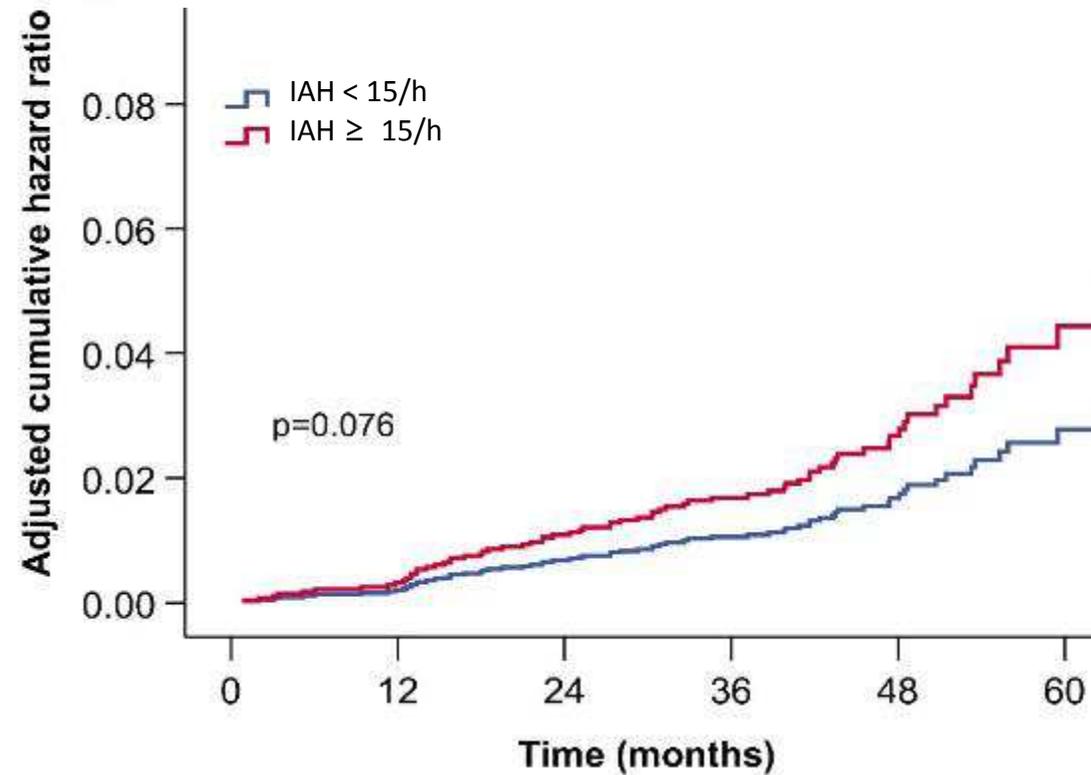
Différence entre IAH <15/h and IAH >15/h (non



Manuel Sánchez-de-la-Torre et al, Lancet resp Med 2019

HynoLaus: population générale

Incidence des événements CV à 5 ans

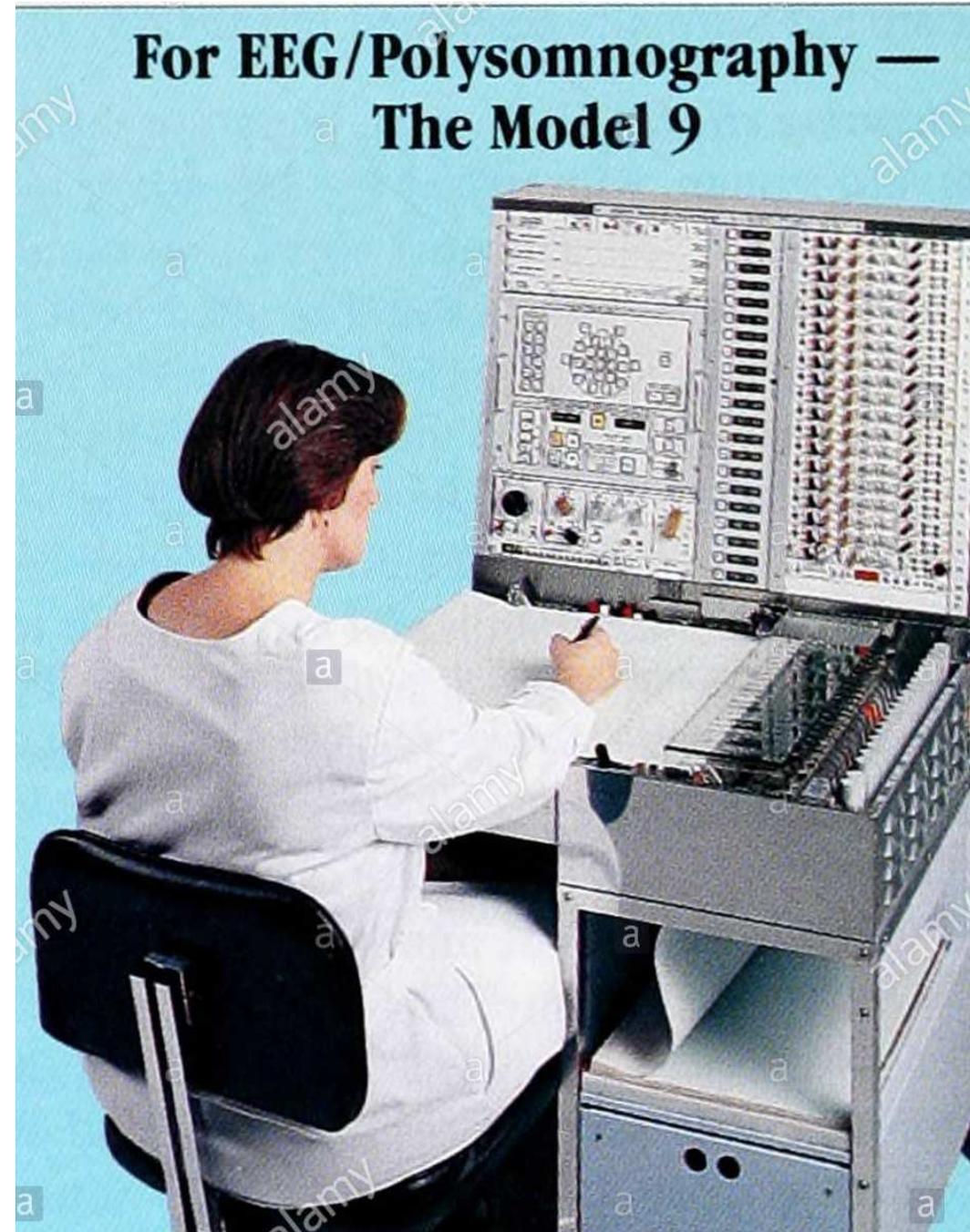
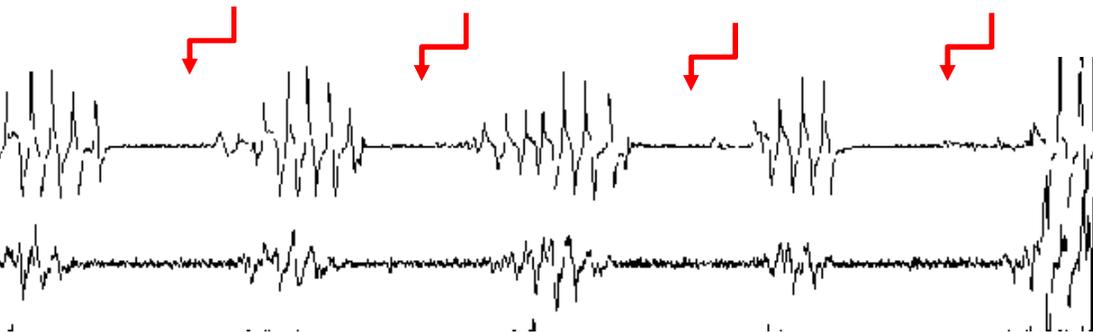


HynoLaus fully adjusted model (unpublished)

**Définition du SAOS selon l'ICSD3 est
essentiellement basée sur l'IAH**

IAH >5/h avec symptômes ou
comorbidités, ou

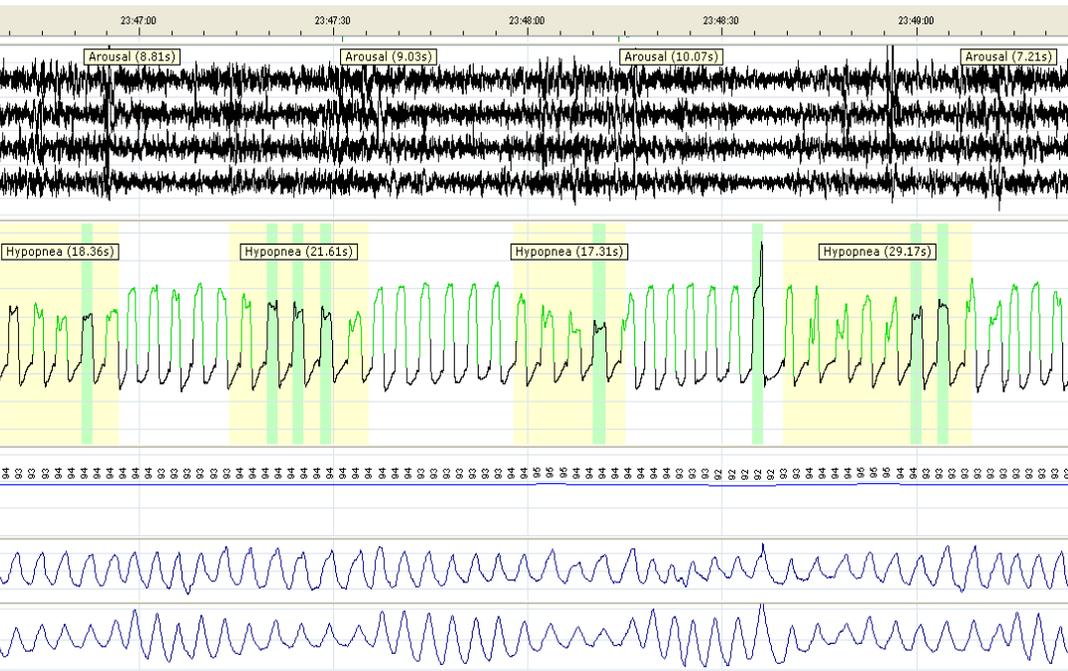
IAH >15/h (même sans symptômes)



The rise and fall of the apnea-hypopnea index: A historical review and critical appraisal

Verzaglie^{1,2}, Barbara Gnidovec-Strazisar³, Ludger Grote⁴, Raphael Heinzer⁵,
McNicholas⁶, Thomas Penzel^{7,8}, Winfried Randerath^{9,10}, Sophia Schiza¹¹,
Braecken¹², Erna S Arnardottir^{13,14}

IAH 45/h

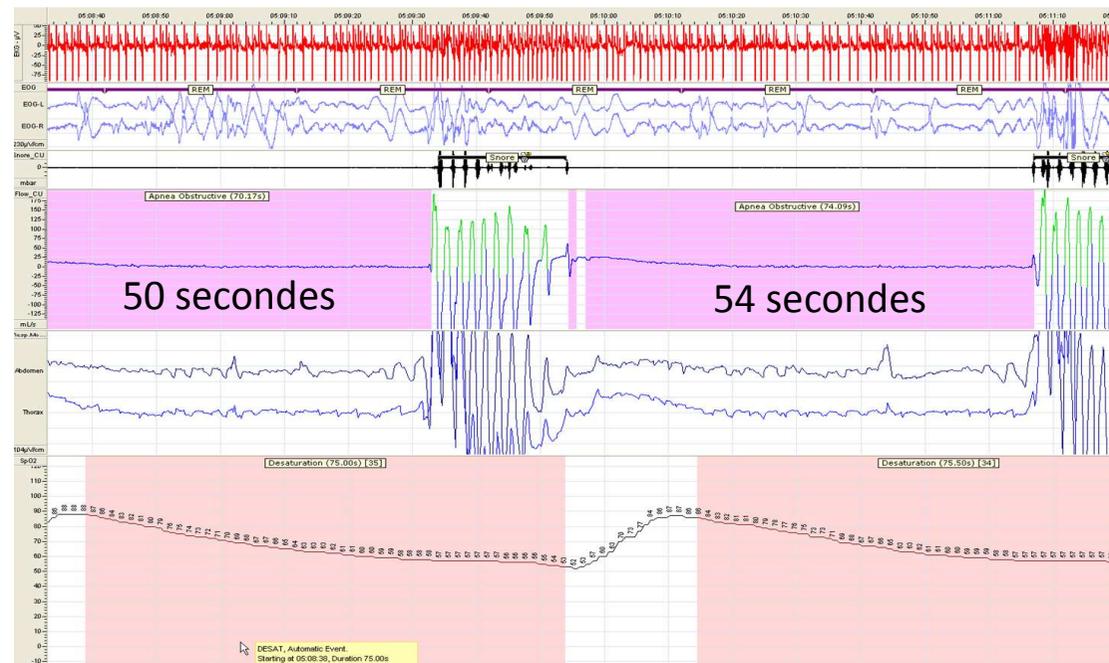


SPECIAL ARTICLE

Metrics of sleep apnea severity: beyond the apnea-hypopnea index

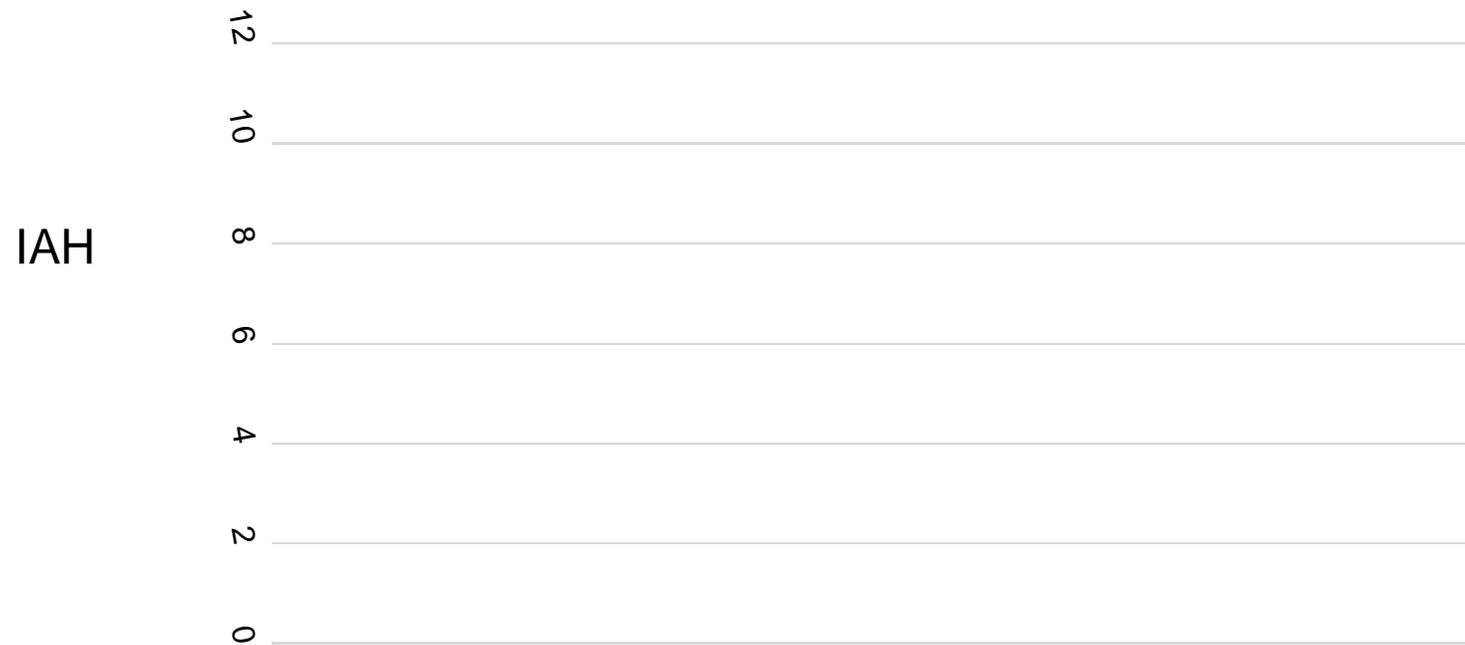
Atul Malhotra^{1,*}, Indu Ayappa², Najib Ayas³, Nancy Collop⁴, Douglas Kirsch⁵,
Nigel Mcardle⁶, Reena Mehra⁷, Allan I. Pack^{8,*}, Naresh Punjabi⁹, David P. White¹⁰,
and Daniel J. Gottlieb^{11,*}—for SRS Task Force

AHI 24/h



IAH dépend des critères de scoring

	Chicago	AASM2007	AASM 2013
Median AHI (/h) (Interquartile range)	10.9 (4.9 – 22.4)	4.4 (1.3 – 11.4)	9.9 (4.2 – 20.1)



N= 2162 PSG

Ce qu'il faut retenir

- . L'IAH seul n'est pas idéal comme critère de sévérité du SAOS
- . Cet index dépend de différents facteurs comme la définition des hypopnées
- . Est-ce qu'il est raisonnable de prendre le même seuil pathologique d'IAH pour une femme de 20 ans et un homme de 80 ans ?
- . Un IAH $>15/h$ ne permet pas de prédire le risque cardiovasculaire

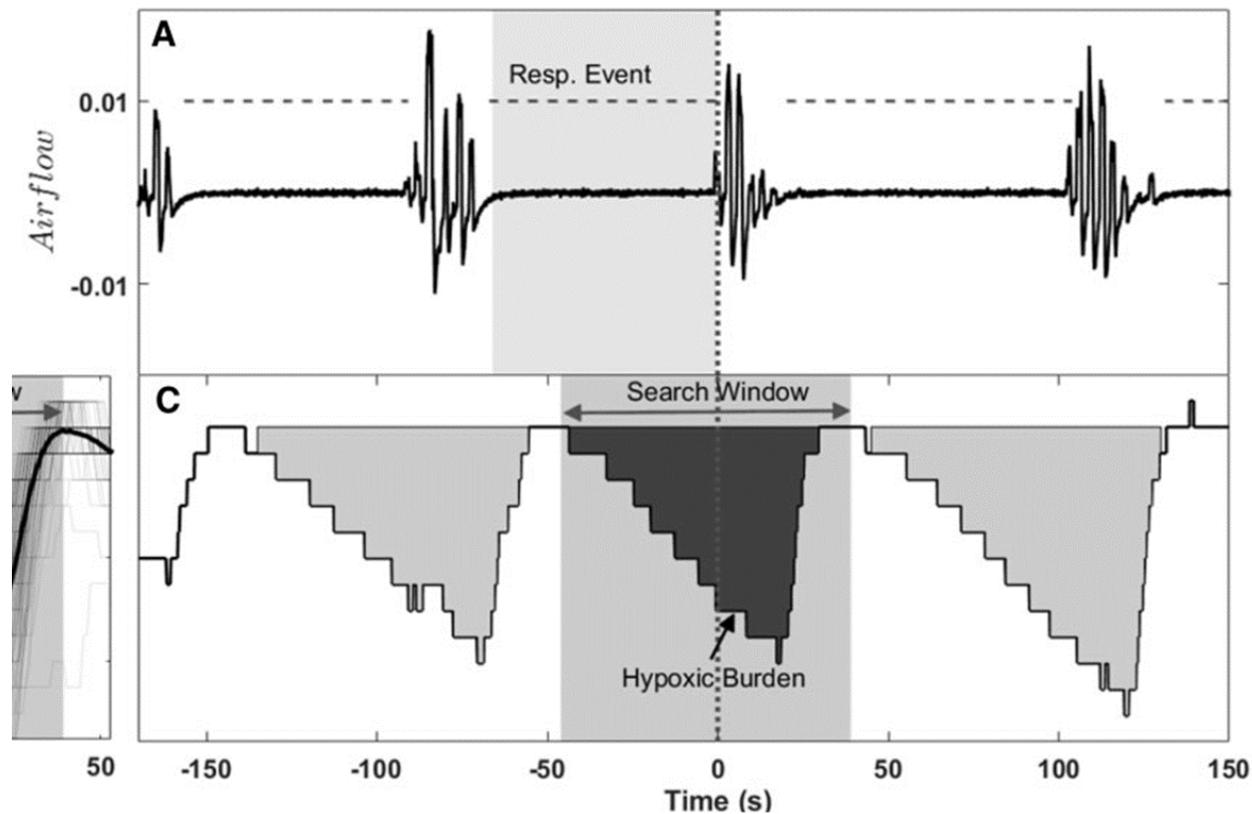
Que suggèrent les cohortes européennes comme autres prédicteurs du risque CV lié au SAOS ?

Hypoxemie ou «charge hypoxique» ?

The **hypoxic burden** of sleep apnoea predicts cardiovascular disease-related mortality: the Osteoporotic Fractures in Men Study and the Sleep Heart Health Study

Ali Azarbarzin^{1*}, Scott A. Sands¹, Katie L. Stone^{2,3}, Luigi Taranto-Montemurro¹, Ludovico Messineo¹, Philip I. Terrill⁴, Sonia Ancoli-Israel^{5,6}, Kristine Ensrud⁷, Shaun Purcell^{1,8}, David P. White¹, Susan Redline¹, and Andrew Wellman¹

European heart journal 20



«Hypoxic Burden»

Somme des “aires sur la courbe” des désaturations en oxygène par heure de sommeil

- Unité: %minute par heure de sommeil
- Par exemple un “hypoxic burden” de 40 (%min)/h
 - = 20 minutes avec desaturation de 2%
 - = 10 minutes de desaturation de 4%
 - = 5 minutes de desaturation de 8%

Hypoxic burden et mortalité cardiovasculaire

Azarbarzin, Eur Heart Journal 2023

MrOS

SHHS

1.00

1.00

1.03 (0.73–1.44)

1.48 (0.93–2.37)

1.01 (0.71–1.42)

1.34 (0.85–2.14)

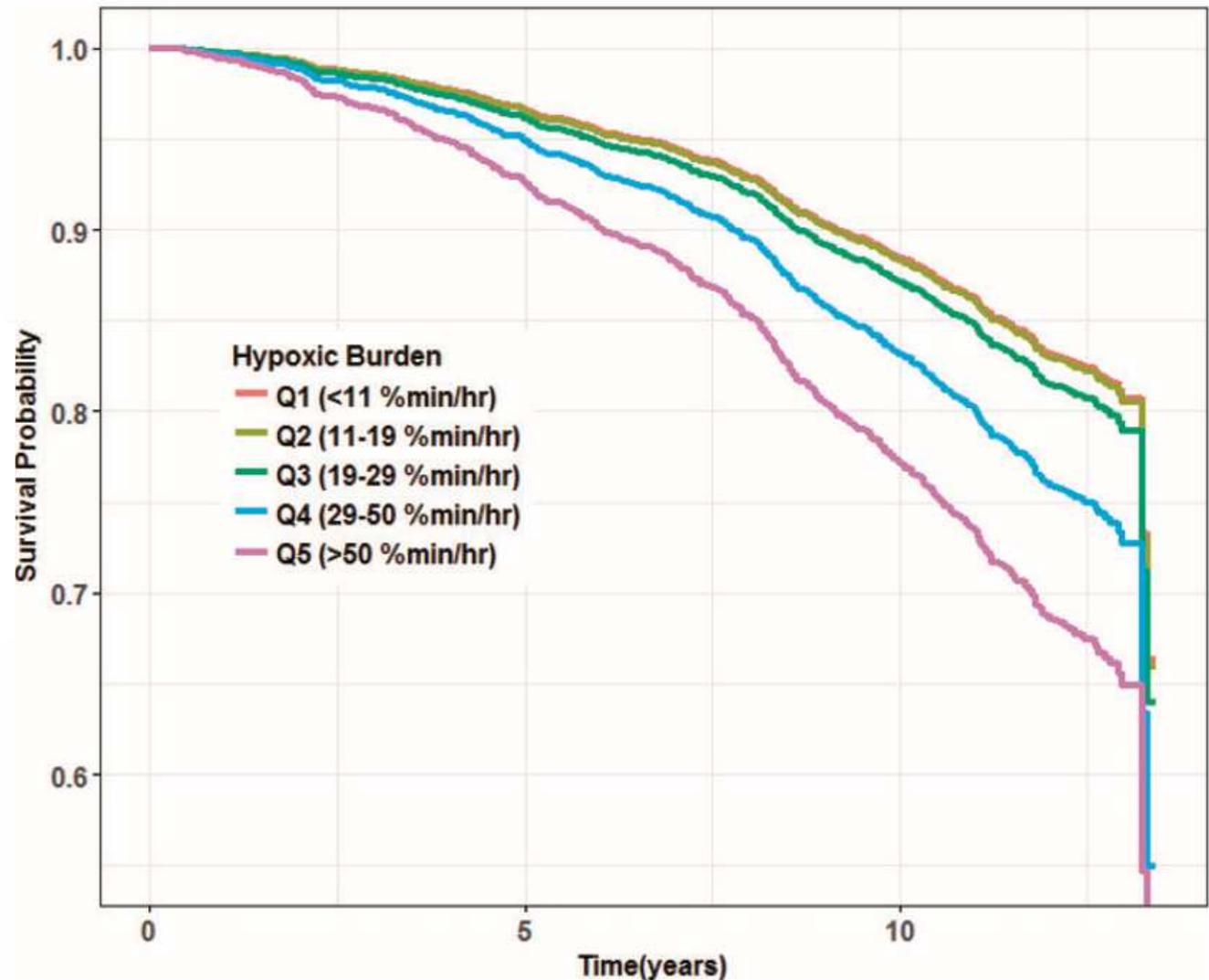
1.60 (1.13–2.28)**

1.61 (1.00–2.61)

2.04 (1.34–3.09)***

1.96 (1.11–3.43)*

Modèle ajusté pour les autres facteurs de risque



Proximal burden and cardiovascular events + all-cause mortality in the Pays de la Loire Sleep Cohort

Trzepizur W. AJRCCM

Pays de La Loire cohort:

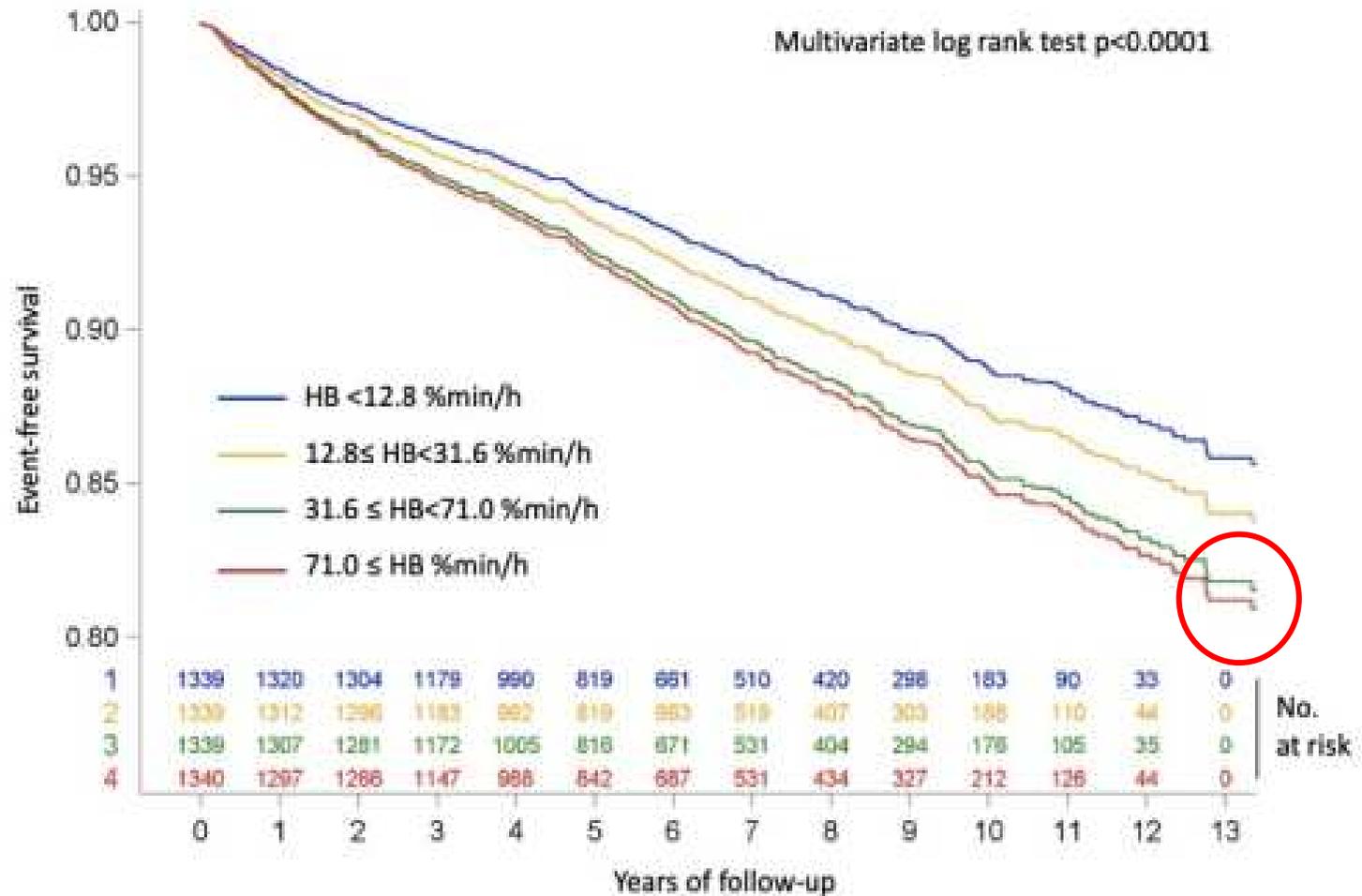
1338 patients, Age 64

(55-73)

Outcome: major adverse

cardiovascular event

(MACE)

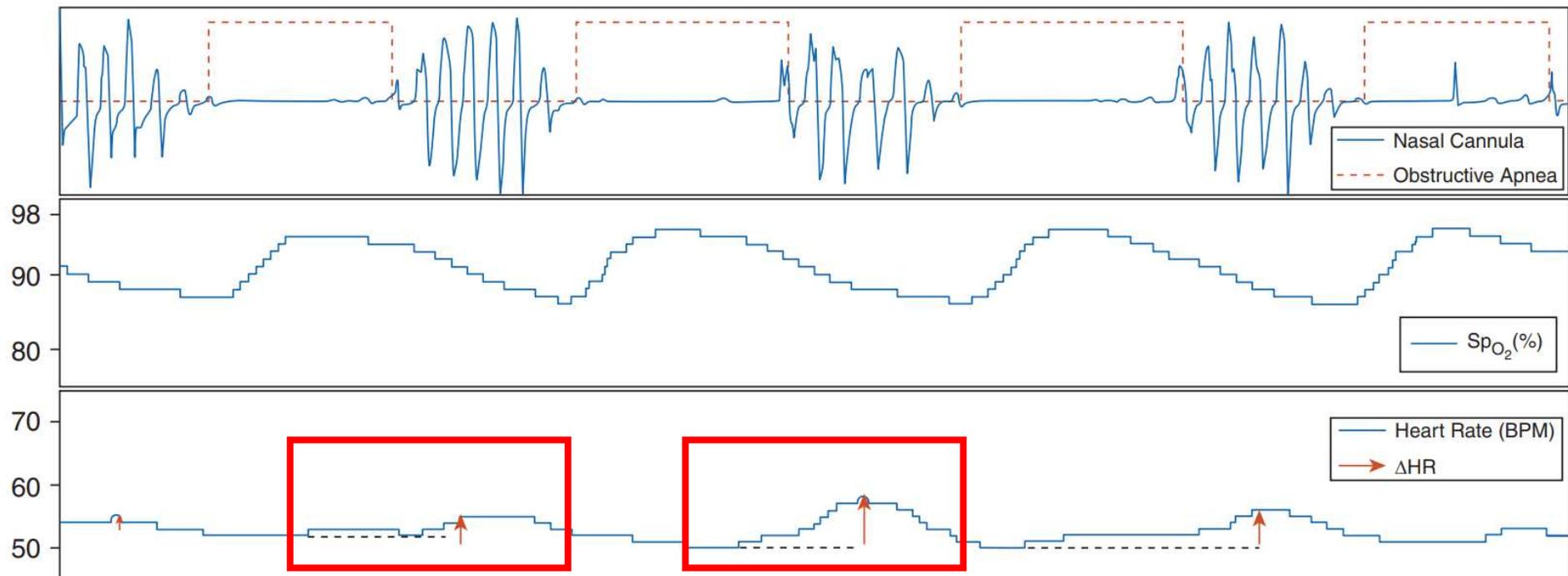


Adjusted for age, gender, body mass index, smoking status, presence of prevalent disease (diabetes, COPD and hypertension), type of sleep study, study site, beta blocker/calcium channel blocker medications and CPAP treatment.

Activations autonomiques pendant le sommeil

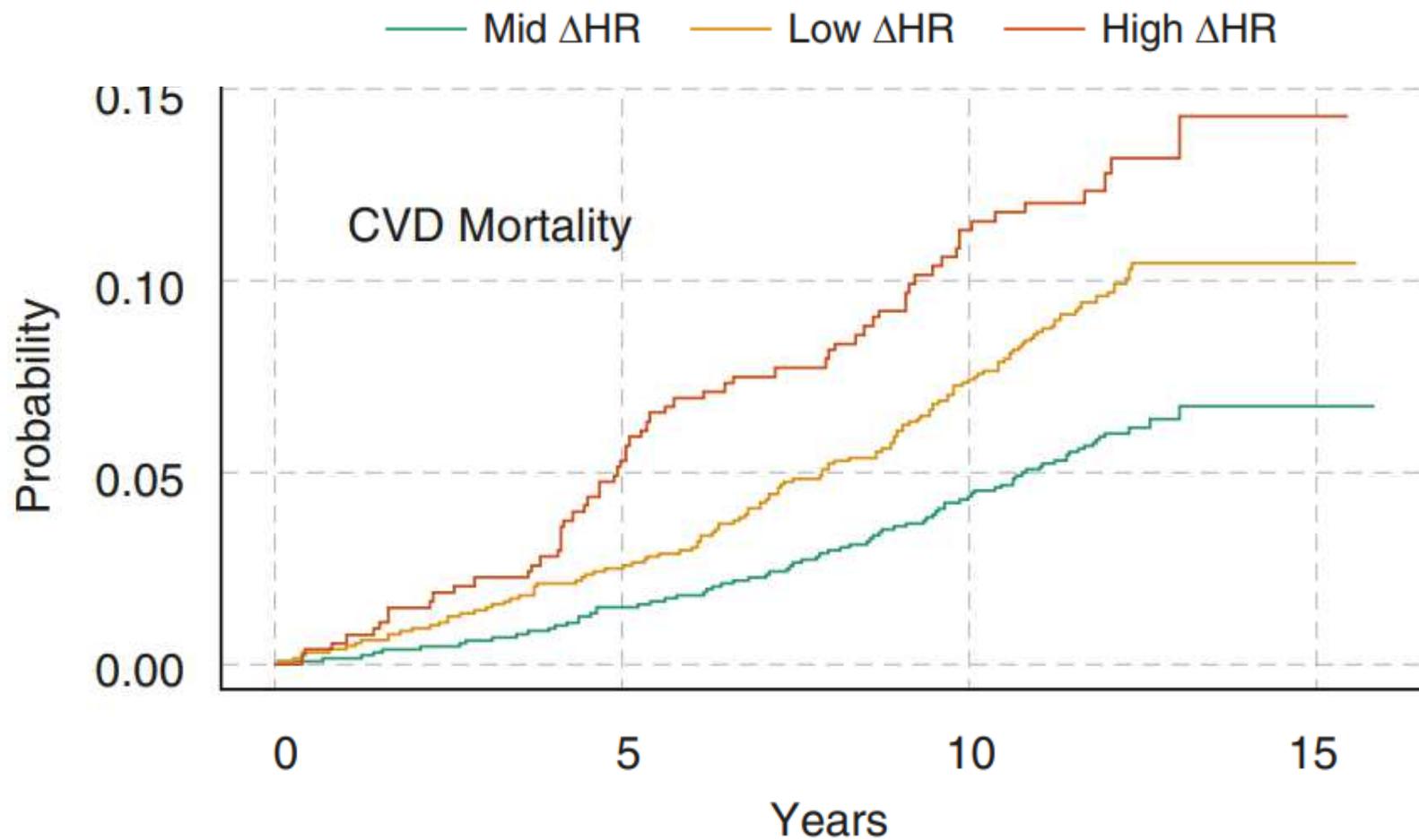
Sleep apnea-specific pulse rate response (Δ heart rate)

Δ HR is the difference between the maximum pulse rate after airway reopening and the minimum pulse rate during respiratory events.



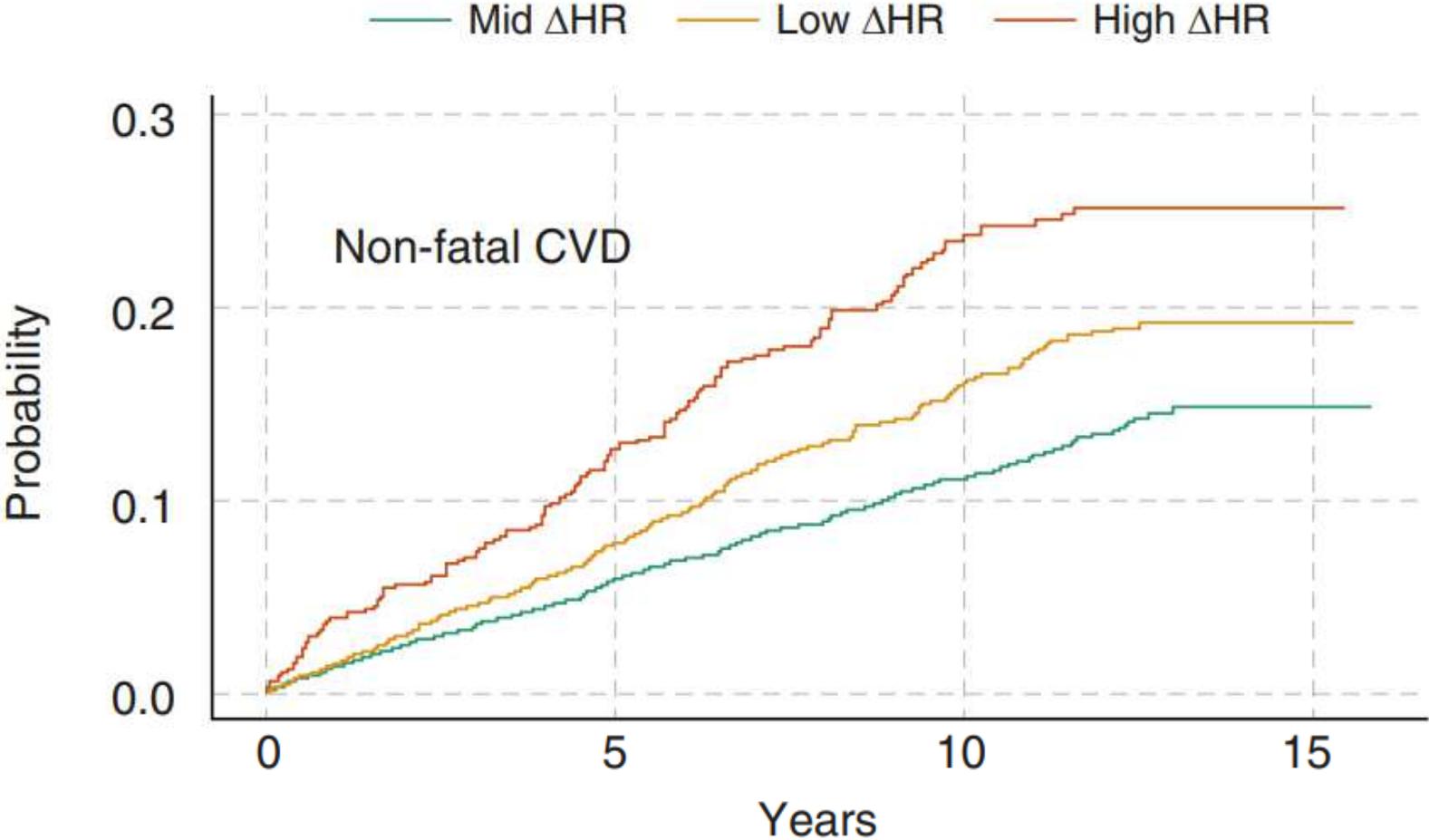
Δ HR and CVD mortality in the Sleep heart health study

Azarbazin A AJRCCM 2021



Δ HR and non fatal CVD in the Sleep heart health study

Azarbazin A AJRCCM 2021

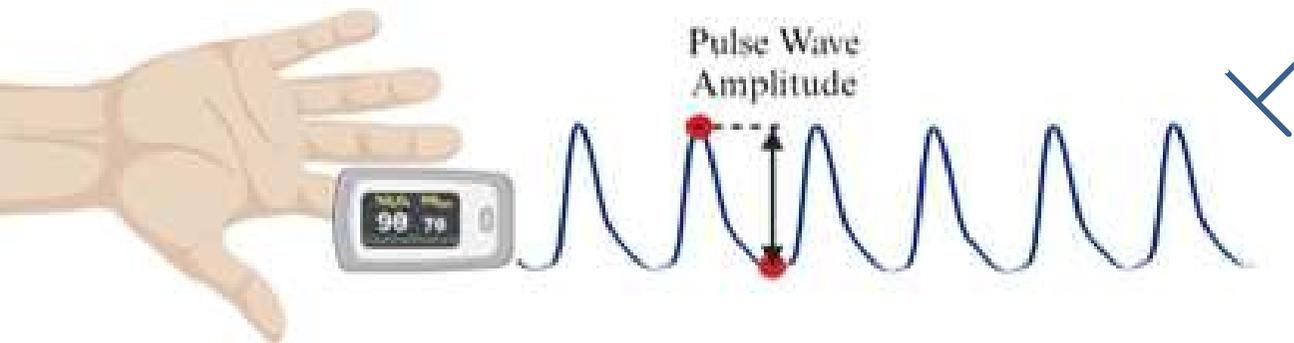
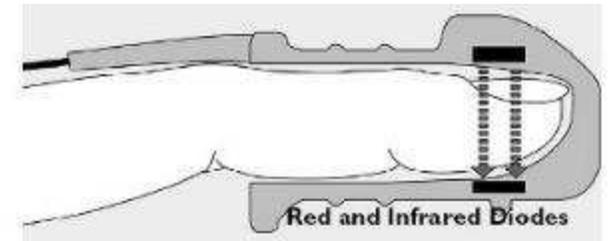


AHR Categories	Nonfatal CVD* [Hazard Ratio (95% CI)]	CVD Mortality [Hazard Ratio (95% CI)]	All-Cause Mortality [Hazard Ratio (95% CI)]
subjects (N = 4,575)			
Midrange	1.00	1.00	1.00
Low	1.07 (0.90–1.27)	1.21 (0.94–1.54)	1.26 (1.10–1.44) [†]
High	1.60 (1.28–2.00)[†]	1.68 (1.22–2.30)[‡]	1.29 (1.07–1.55)[‡]
II ≥ 15 events/h (N = 2,148)			
Midrange	1.00	1.00	1.00
Low	1.13 (0.90–1.43)	1.39 (1.00–1.93)	1.13 (0.94–1.36)
High	1.69 (1.28–2.22)[†]	1.92 (1.29–2.86)[‡]	1.36 (1.09–1.71)[‡]
II < 15 events/h (N = 2,427)			
Midrange	1.00	1.00	1.00
Low	0.97 (0.75–1.25)	0.98 (0.67–1.42)	1.40 (1.14–1.71)[‡]
High	1.60 (1.06–2.39)[§]	1.28 (0.73–2.22)	1.08 (0.75–1.55)
II ≥ 30 events/h (N = 820)			
Midrange	1.00	1.00	1.00
Low	1.40 (0.95–2.04)	2.09 (1.17–3.74)[§]	1.11 (0.82–1.51)
High	1.90 (1.28–2.80)[‡]	2.96 (1.60–5.48)[†]	1.51 (1.10–2.07)[§]
II < 30 events/h (N = 3,755)			
Midrange	1.00	1.00	1.00
Low	1.01 (0.83–1.23)	1.06 (0.81–1.39)	1.29 (1.11–1.50)[†]
High	1.51 (1.13–2.00)[‡]	1.38 (0.93–2.03)	1.16 (0.91–1.48)
III ≥ 62%min/h (N = 1,138)			
Midrange	1.00	1.00	1.00
Low	1.38 (1.00–1.89)[§]	1.89 (1.19–3.00)[‡]	1.44 (1.13–1.83)[‡]
High	1.93 (1.36–2.73)[†]	3.50 (2.15–5.71)[†]	1.84 (1.40–2.40)[†]
III < 62%min/h (N = 3,409)			
Midrange	1.00	1.00	1.00
Low	0.96 (0.78–1.17)	0.94 (0.70–1.26)	1.17 (1.00–1.38)
High	1.35 (0.99–1.84)	0.93 (0.58–1.50)	0.89 (0.66–1.18)

Pulse wave amplitude drops (PWAD)

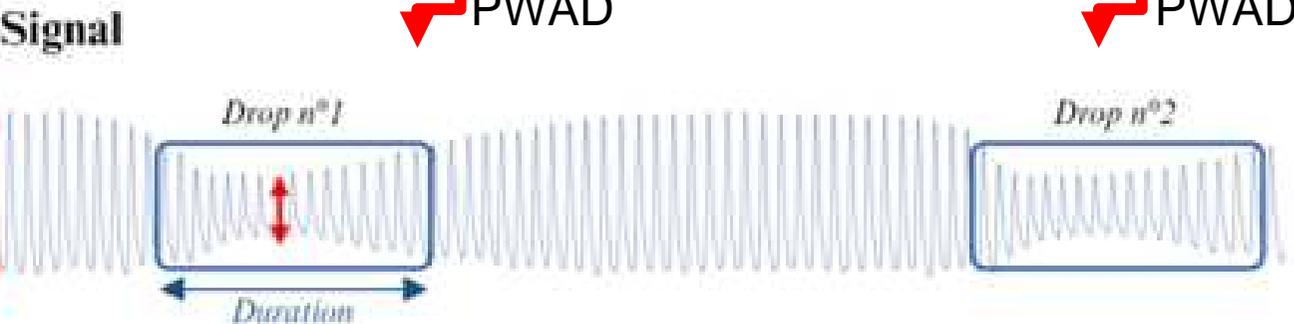
Pulse wave amplitude (PWA) can be derived from the photoplethysmography signal (PPG) of the pulseoximeter

Pulse oximeter

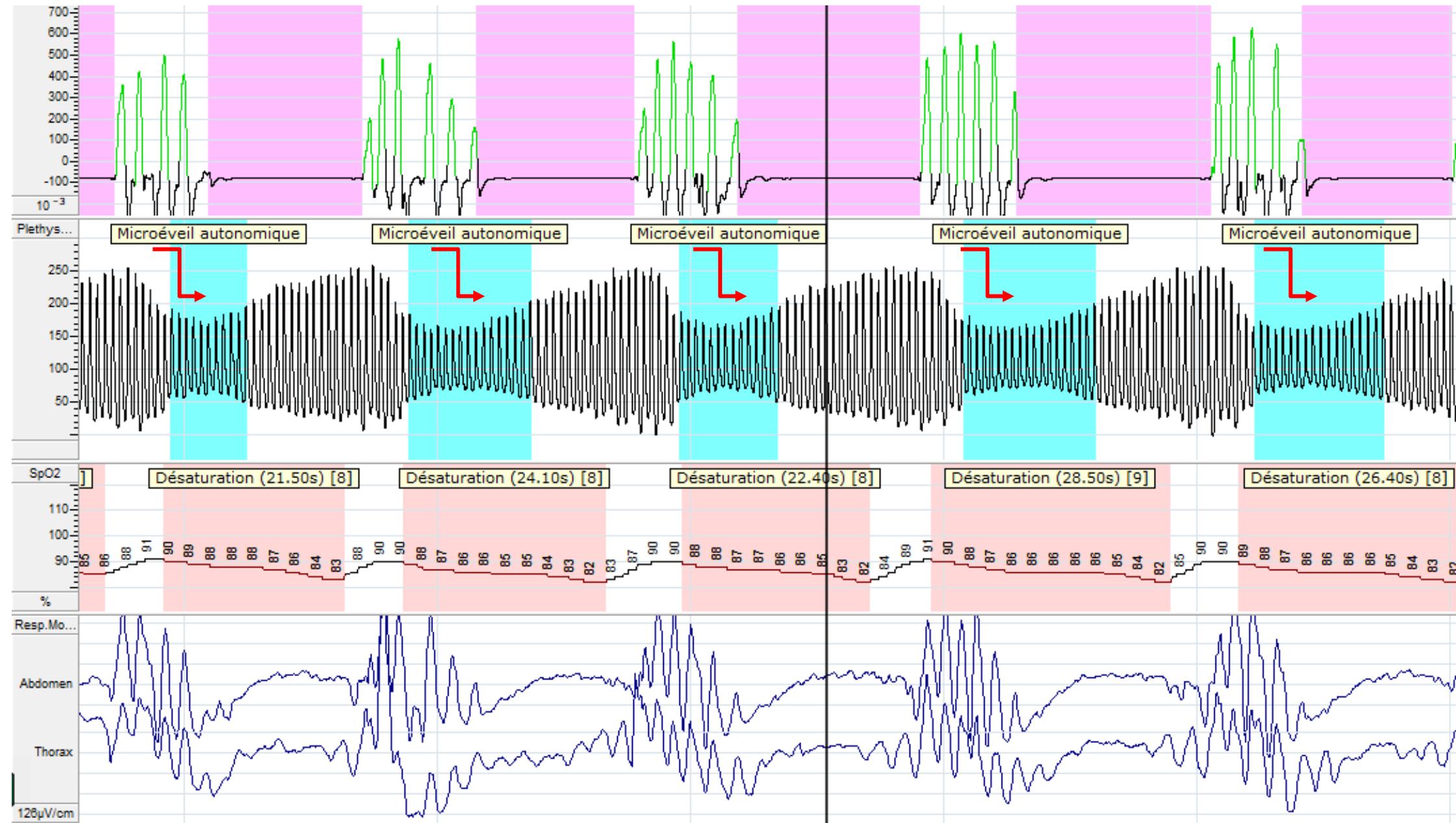


➤ PWA signal is related to variation of the tissue blood volume

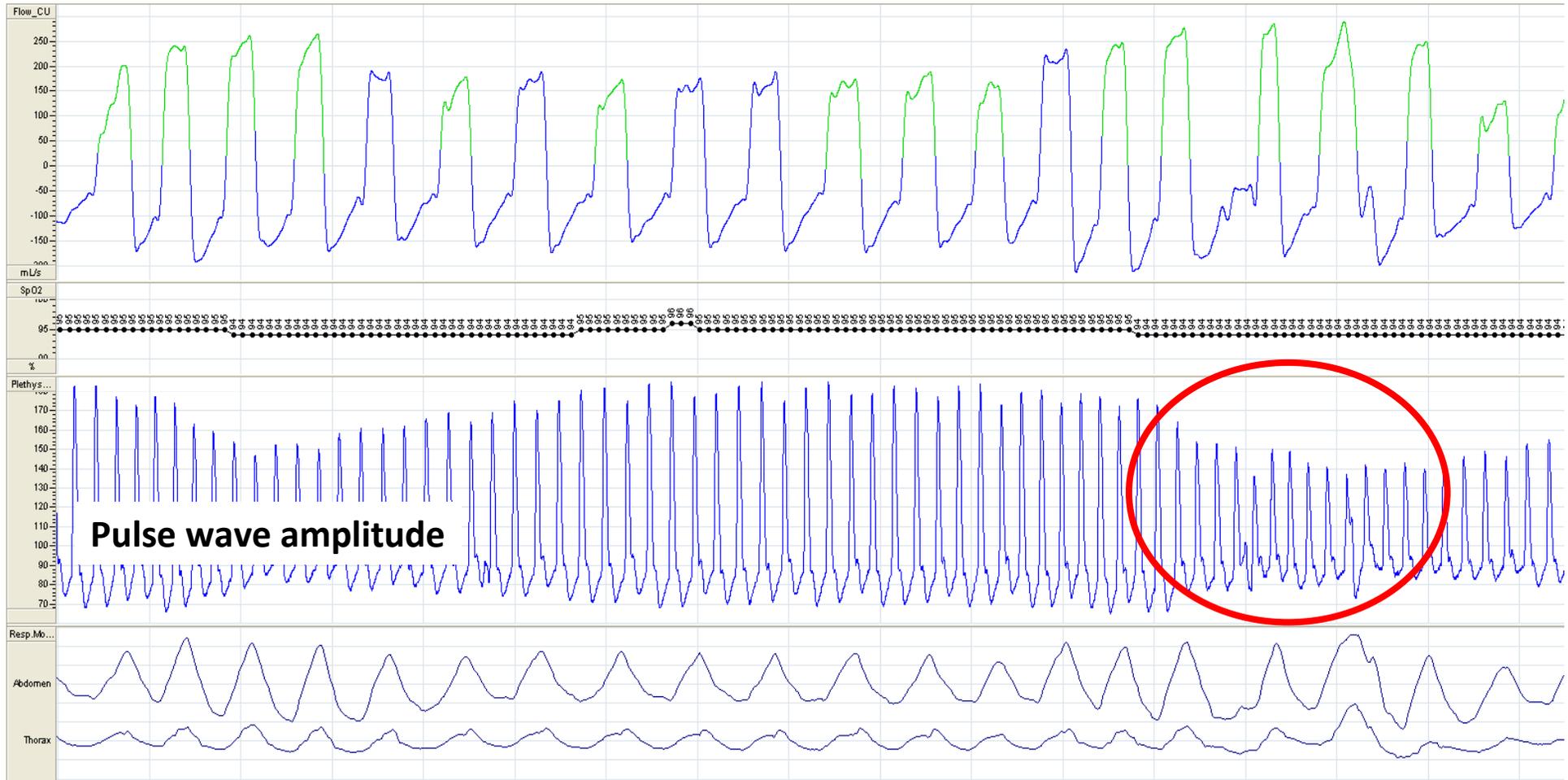
➤ PWA drops (PWAD) reflect peripheral vasoconstriction resulting from sympathetic activation

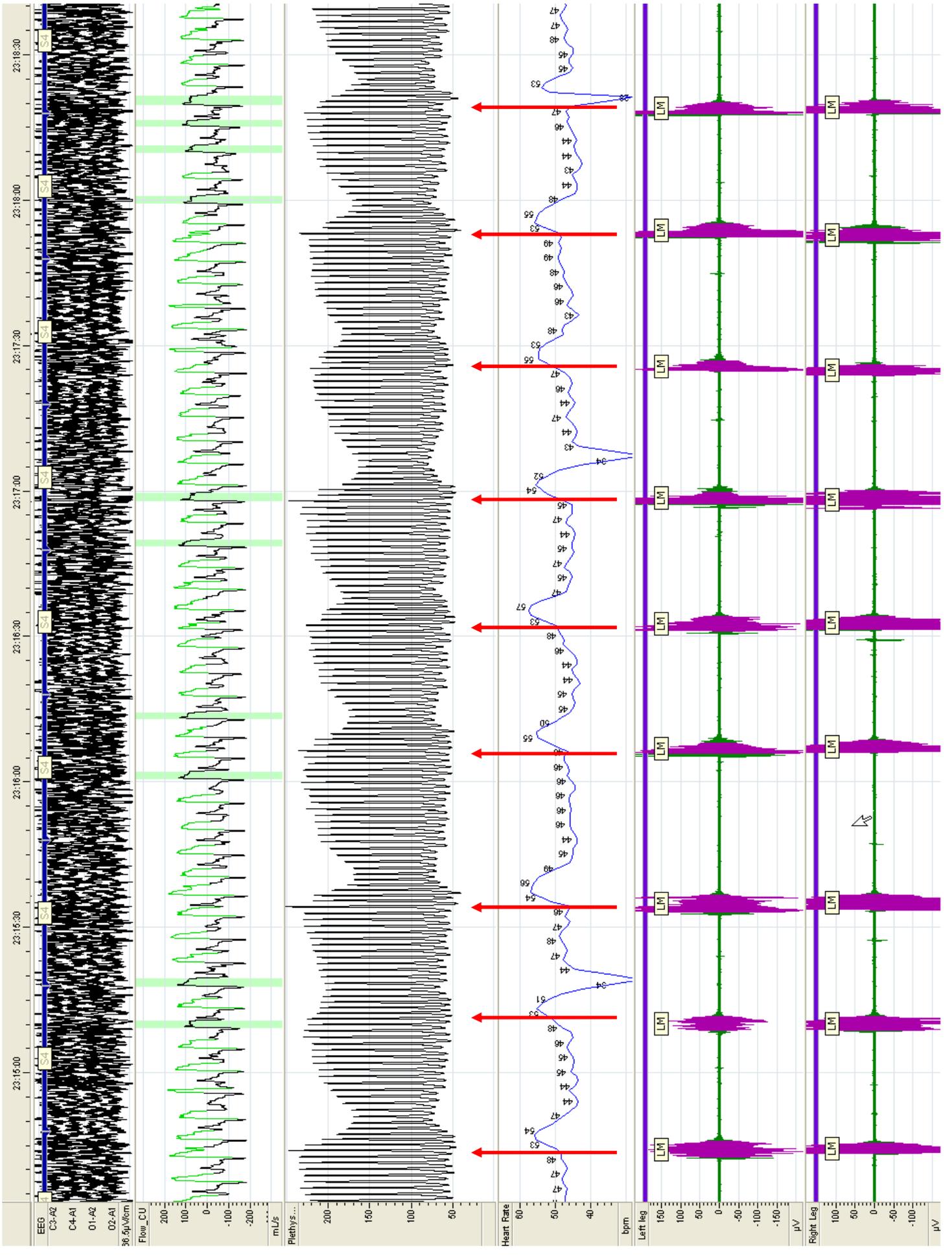


PWADs can occur after respiratory events

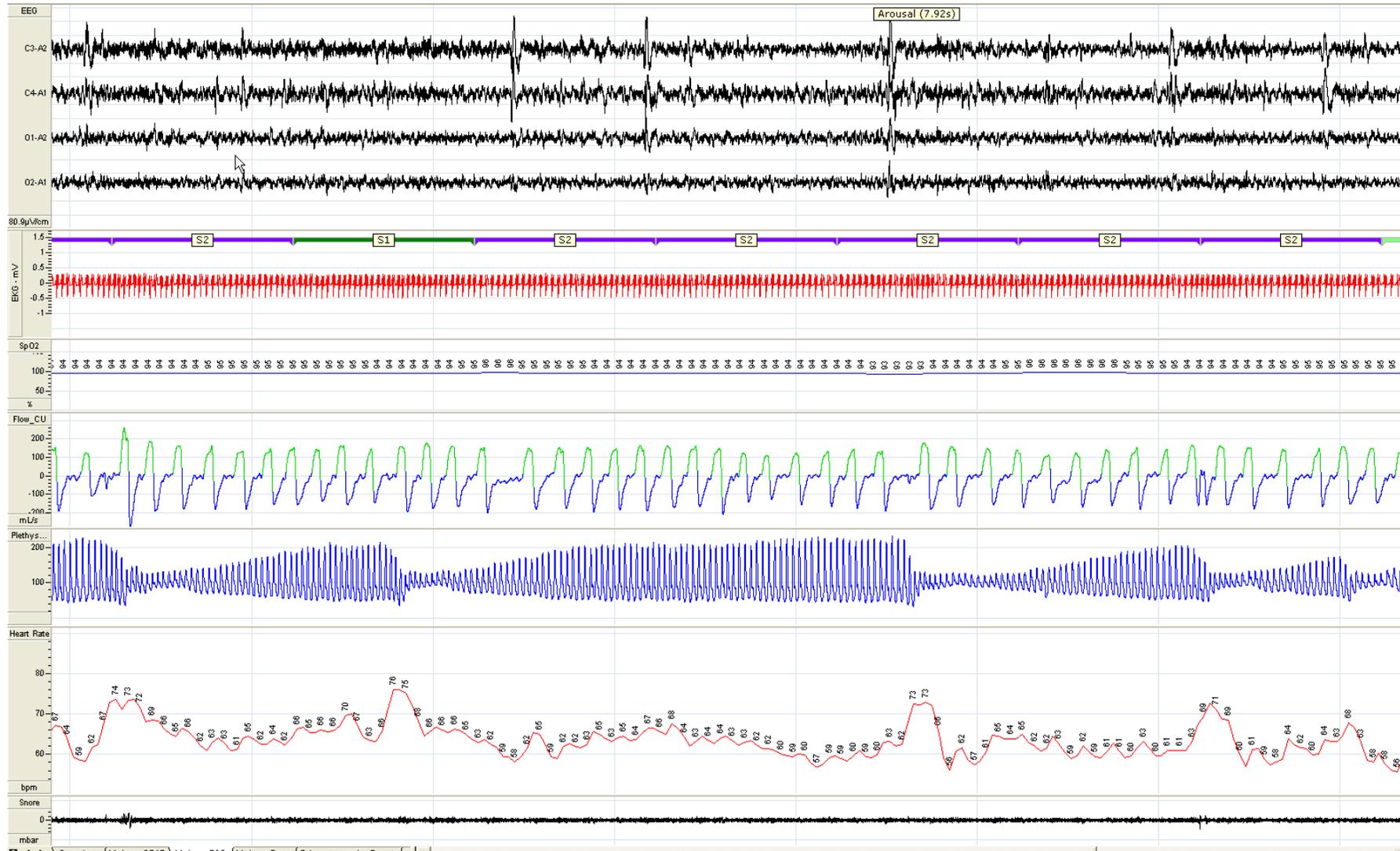


RERA and PWA drop





PWAD espontáneos



PWAD index:

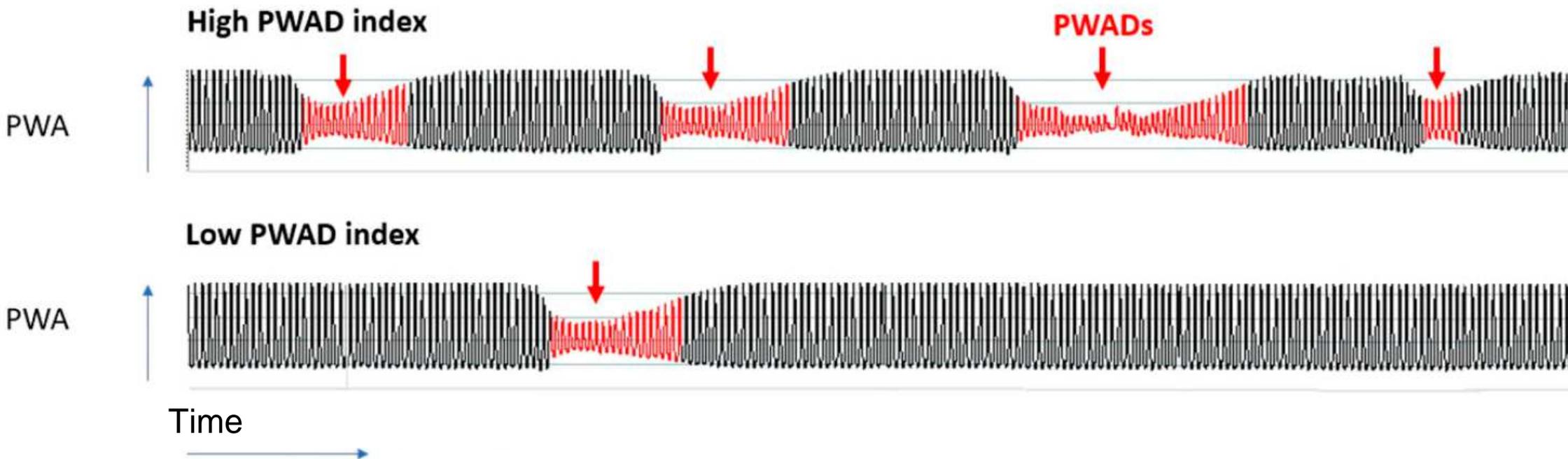
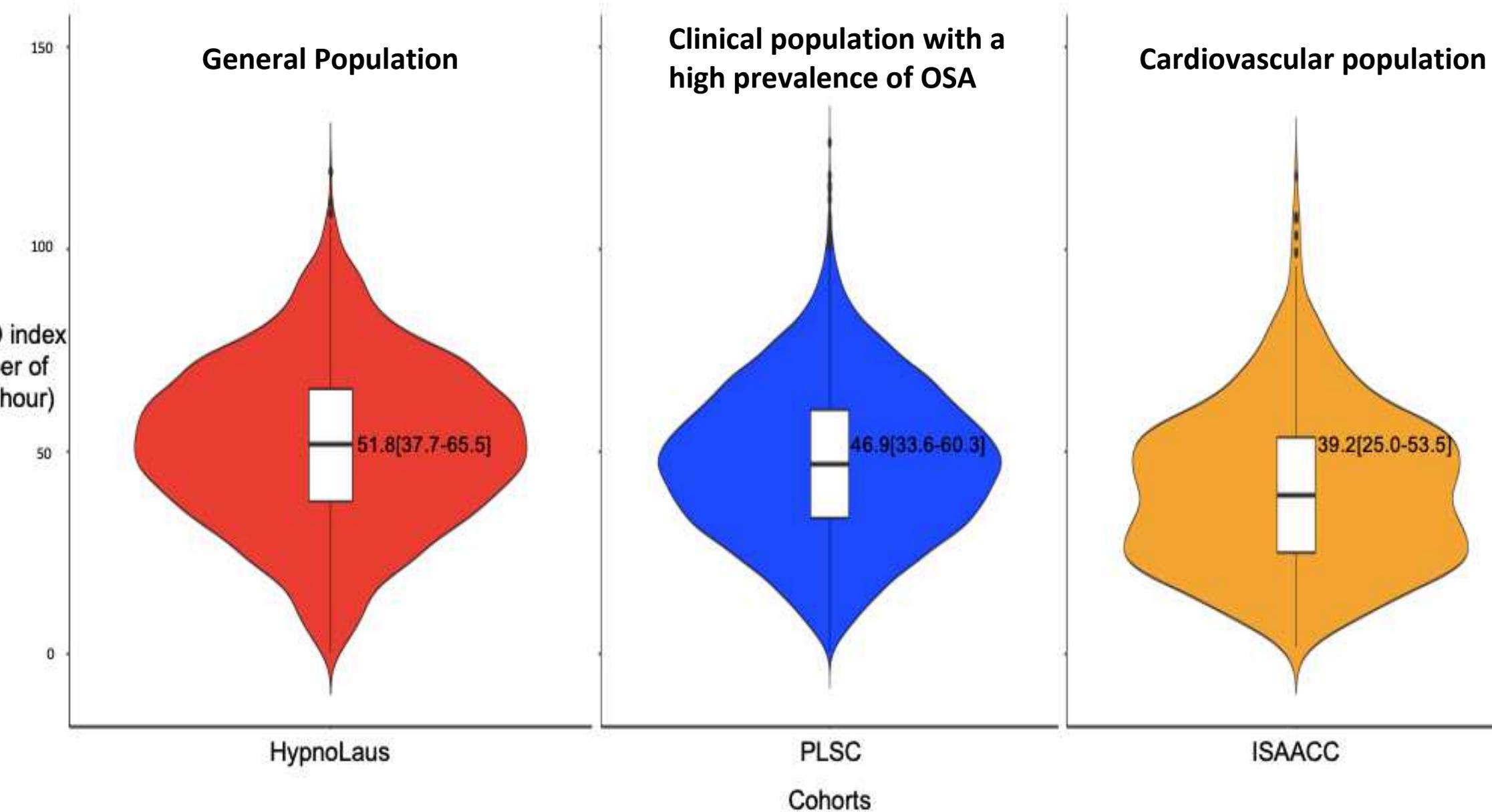
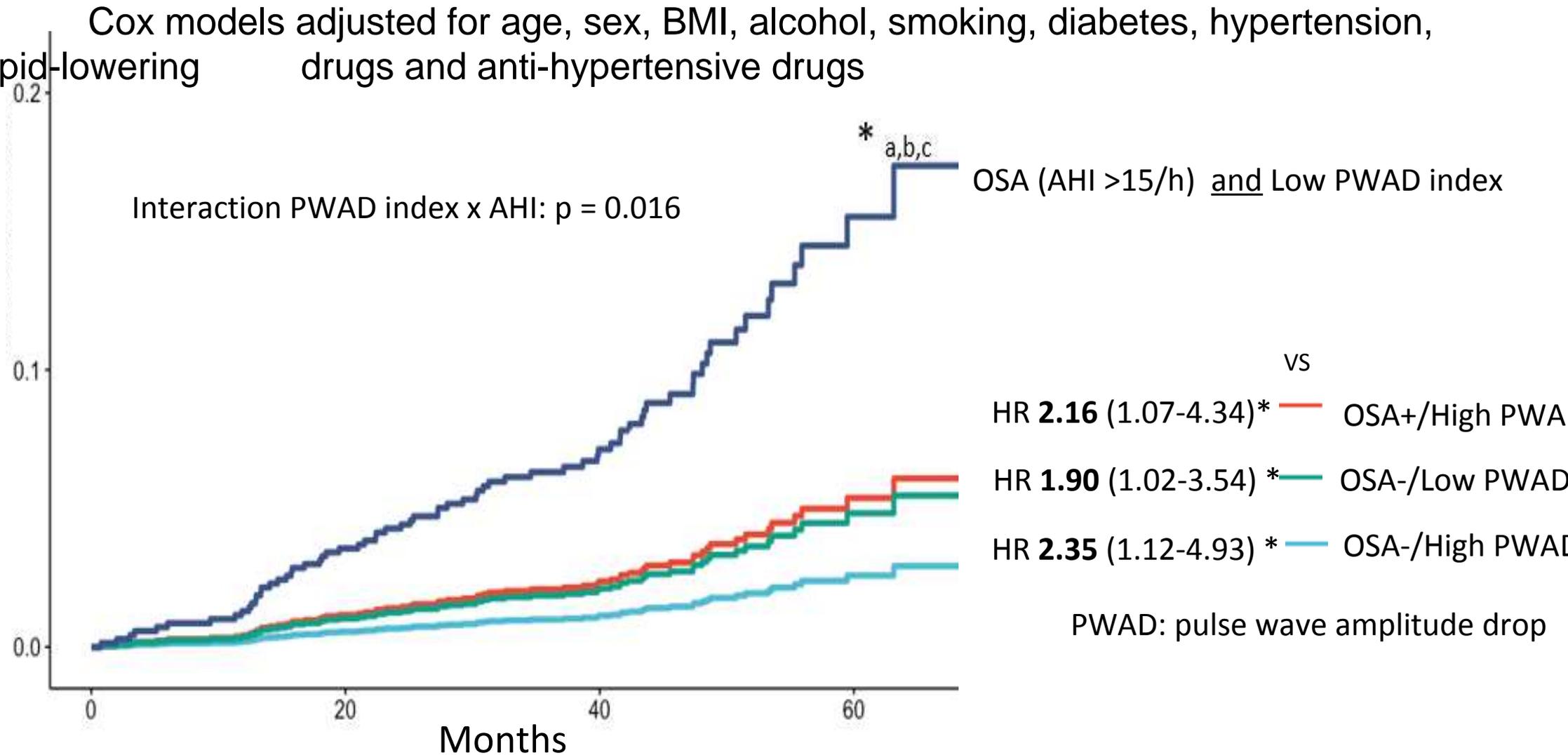


Figure 1. PPG and PWA assessment. PPG = photoplethysmography; PWA = pulse wave amplitude; PWAD = pulse wave amplitude drop.

PWAD index in different populations

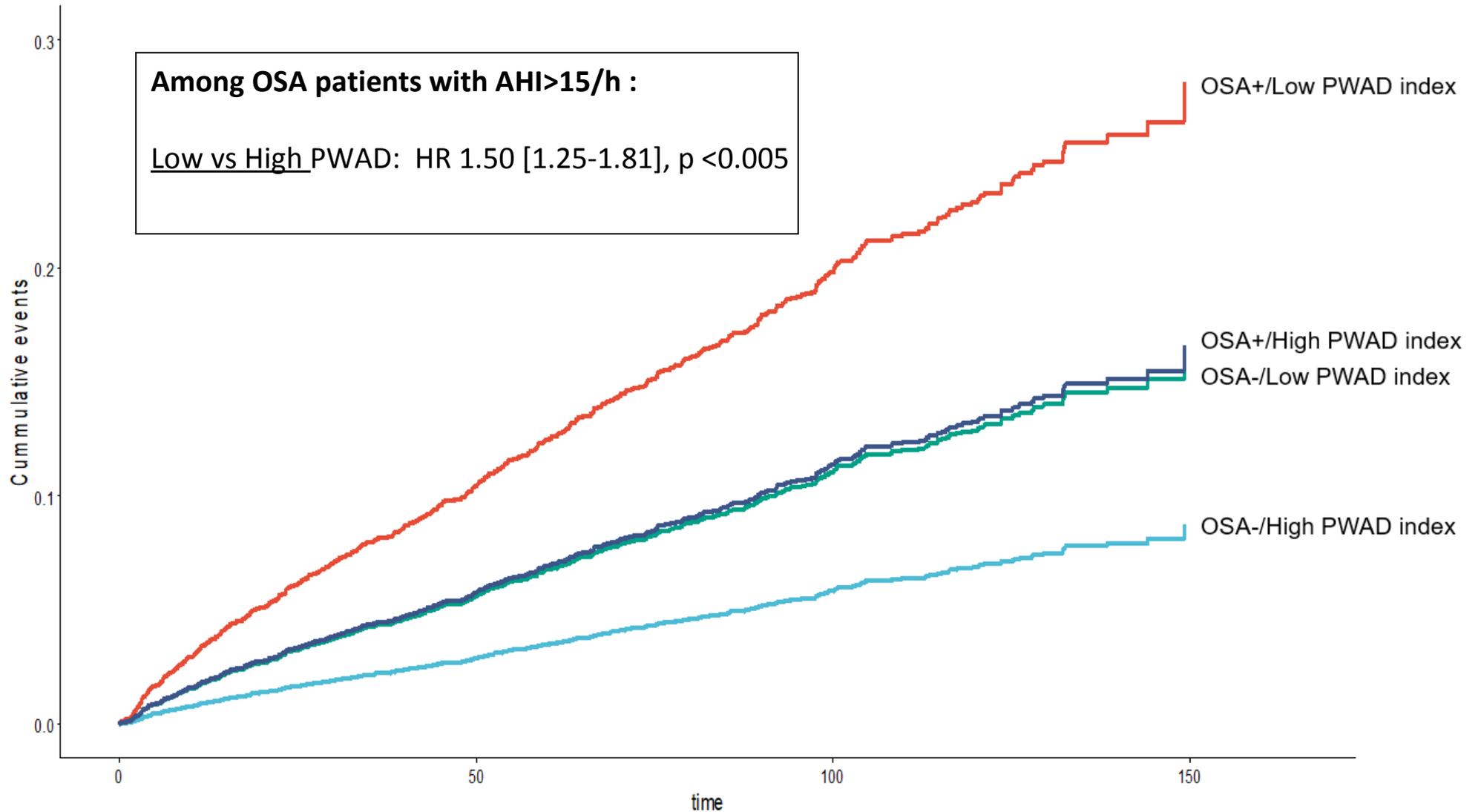


HypoLaus cohort



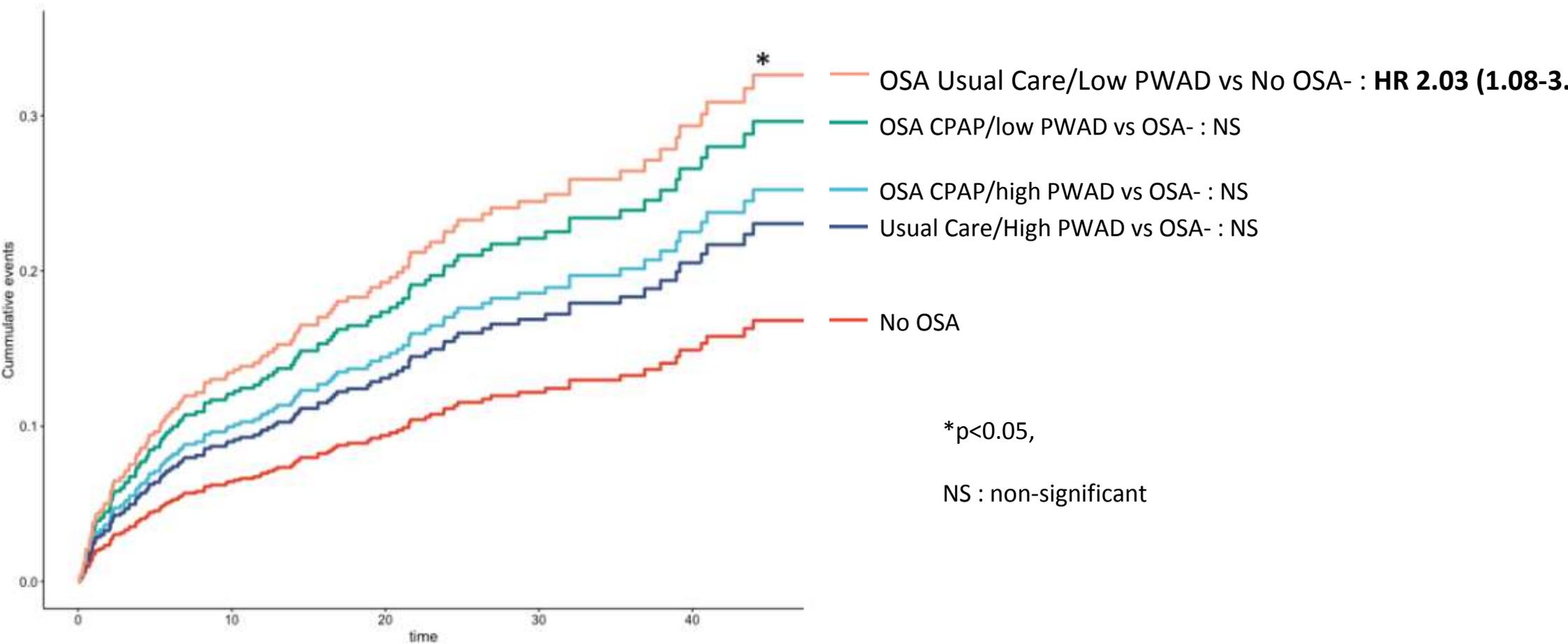
Pays de la Loire cohort (PLSC): incidence of MACE

Adjusted for age, gender, BMI, diabetes, hypertension, smoking, site, recording type (PG/PSG), vasodilators, CP



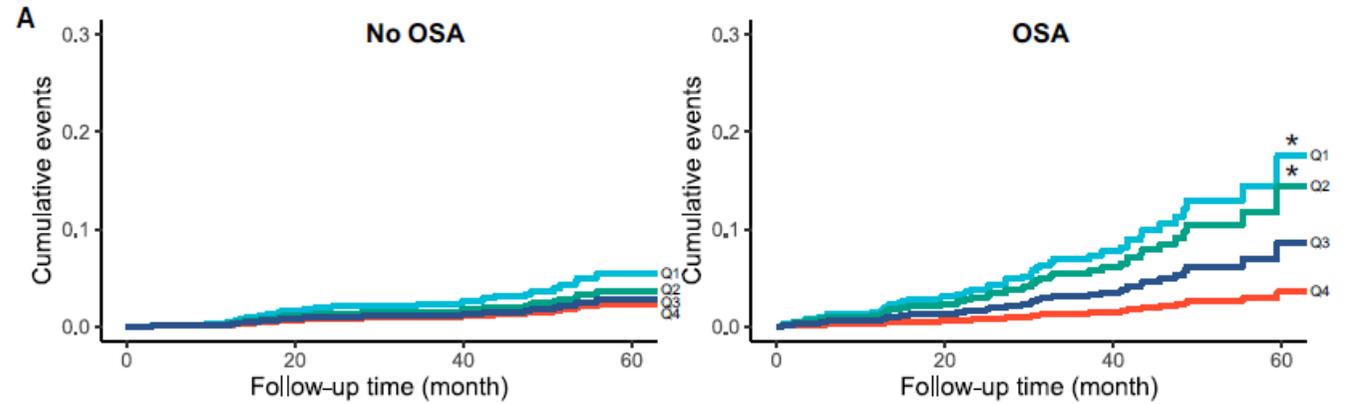
ISAACC cohort: recurrence of CV events

Adjusted for age, sex, body mass index, alcohol, smoking, diabetes, hypertension, lipid-lowering drugs, B-blockers

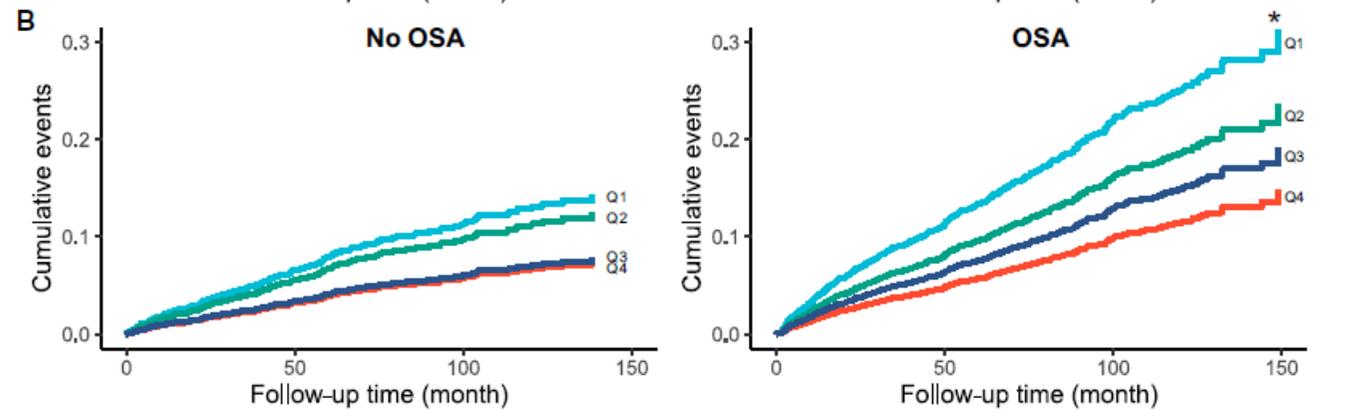


PWAD appears to be a CV risk marker specific to OSA

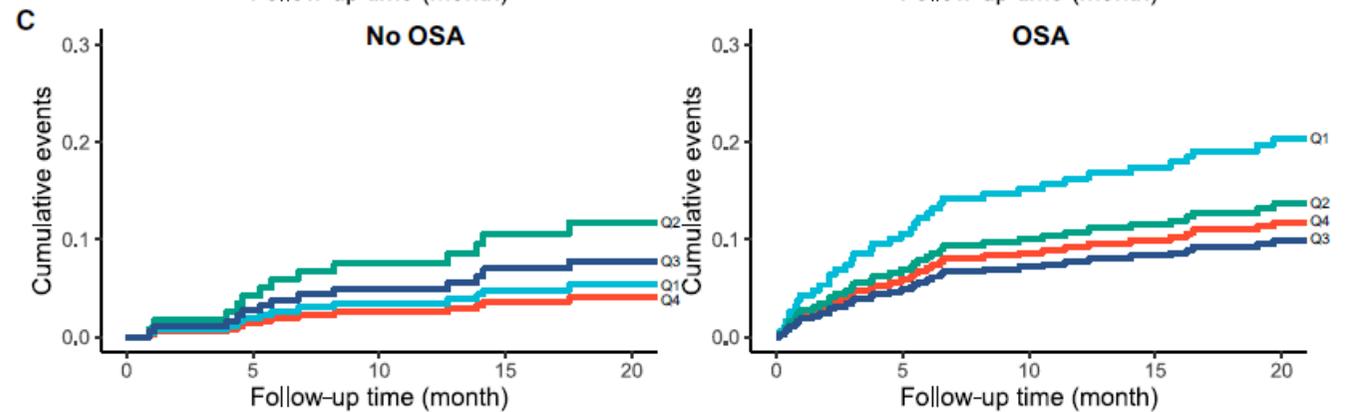
HypnoLaus



Pays de la Loire

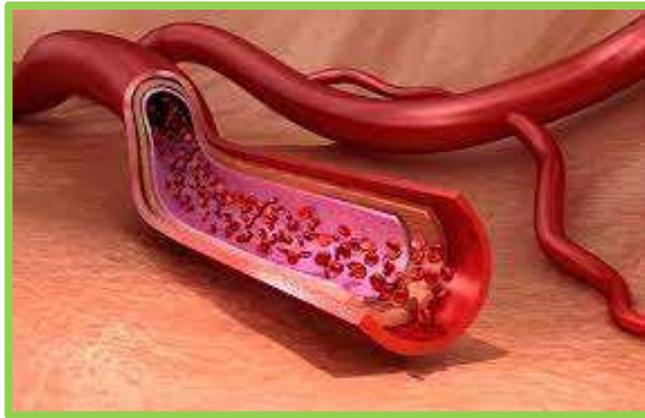


ISAACC

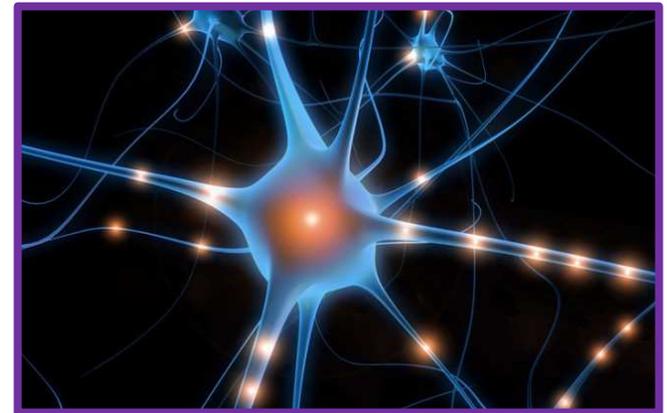
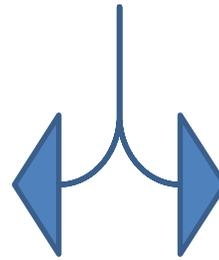


PWAD index and cardiovascular risk

Low pulse wave amplitude drop index



Endothelial dysfunction



Blunted autonomic reactivity



Increased cardiovascular risk



Hypothèses

Un PWAD index bas dans le SAOS pourrait être dû à :

1. Un **système nerveux autonome** peu réactif du à un émoussement des barorécepteurs
1. Une **fonction endothéliale altérée**, avec une moindre capacité des artères et des capillaires à se contracter et à se dilater rapidement.

Est-ce la PPC permet de réduire la mortalité et le risque cardiovasculaire ?

Relationship Between CPAP Termination and All-Cause Mortality

French Nationwide Database Analysis

Pépin et al Chest 2022

*Louis Pépin, MD; Sébastien Bailly, PhD; Pierre Rinder, MSc; Dan Adler, MD; Adam V. Benjafield, PhD;
Antoine Lavergne, MSc; Anne Josseran, PharmD; Paul Sinel-Boucher, MSc; Renaud Tamisier, MD; Peter A. Cistulli, MD;
Vishal Malhotra, MD; Pierre Hornus, MSc; on behalf of the medXcloud Group*

Etude basée sur le Système National des Données de Santé (SNDS) français

Deux groupes pairés (arrêt vs poursuite PPC) incluant chacun 88,007 patients

Age moyen 60 ans, 65% hommes

Outcome primaire: mortalité

Propensity score matching

Relationship Between CPAP Termination and All-Cause Mortality

A French Nationwide Database Analysis

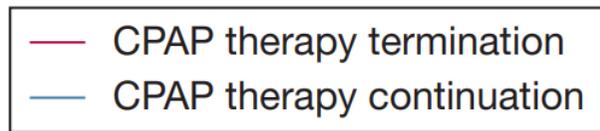
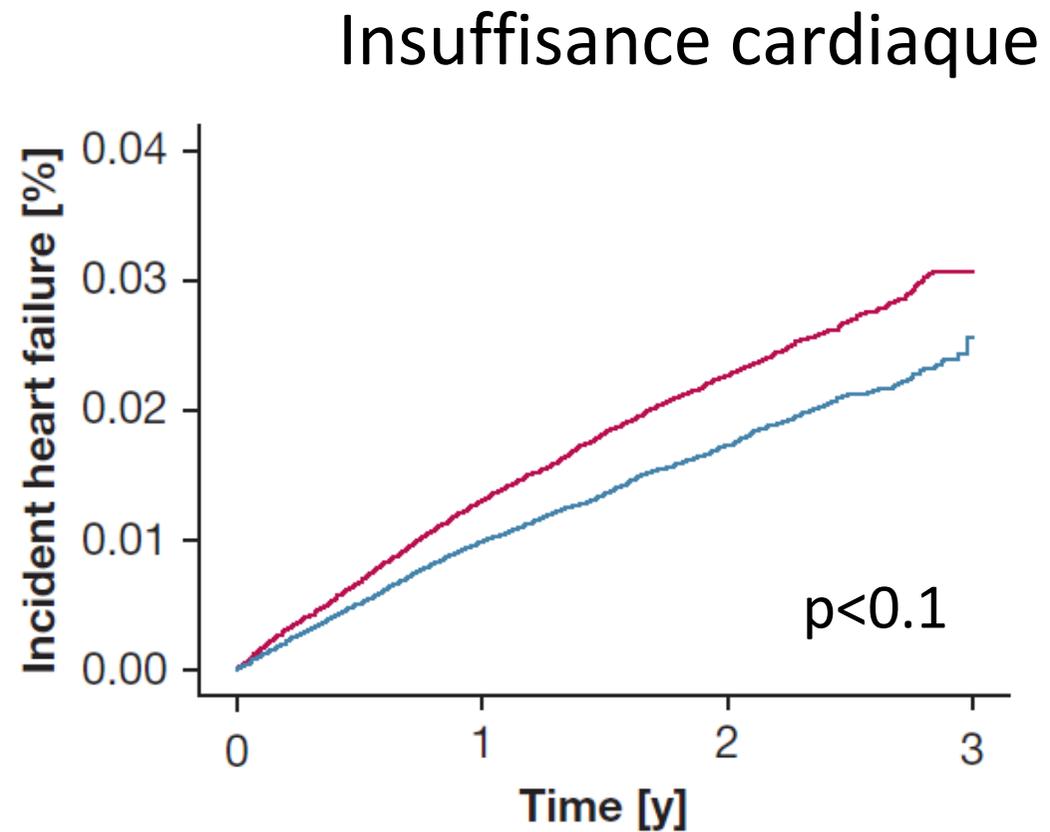
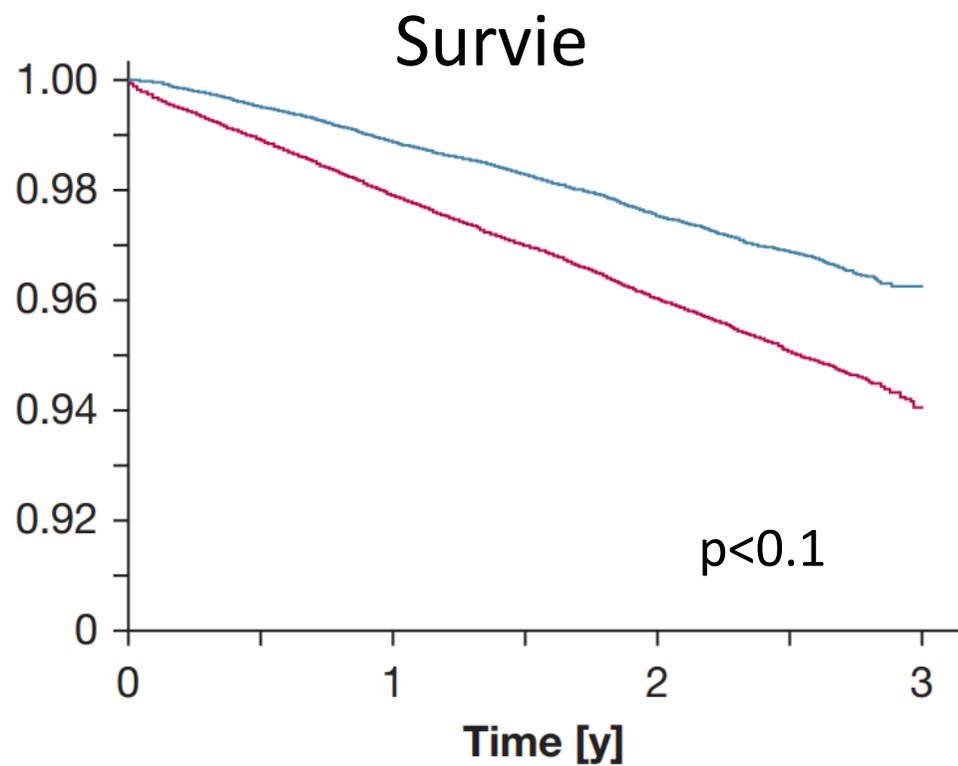
Pépin JL. Chest 2022

TABLE 1] Baseline Characteristics of the Matched Study Population

Variable	CPAP Continuation (n = 88,007)	CPAP Termination (n = 88,007)
Age, y	60.0 (70.0-50.0)	59.0 (69.0-49.0)
Female sex	32,227 (36.6)	31,666 (36.0)
Comorbidity		
Chronic psychiatric disorders	4,621 (5.2)	4,606 (5.2)
Stroke	2,735 (3.1)	2,684 (3.1)
Heart failure	2,306 (2.6)	2,046 (2.3)
Coronary heart disease	8,023 (9.1)	8,037 (9.1)
Hypertension	42,568 (48.4)	43,231 (49.1)
Diabetes mellitus	18,610 (21.1)	18,304 (20.8)
COPD	7,156 (8.1)	7,387 (8.4)

Data are presented as No. (%) or median (interquartile range).

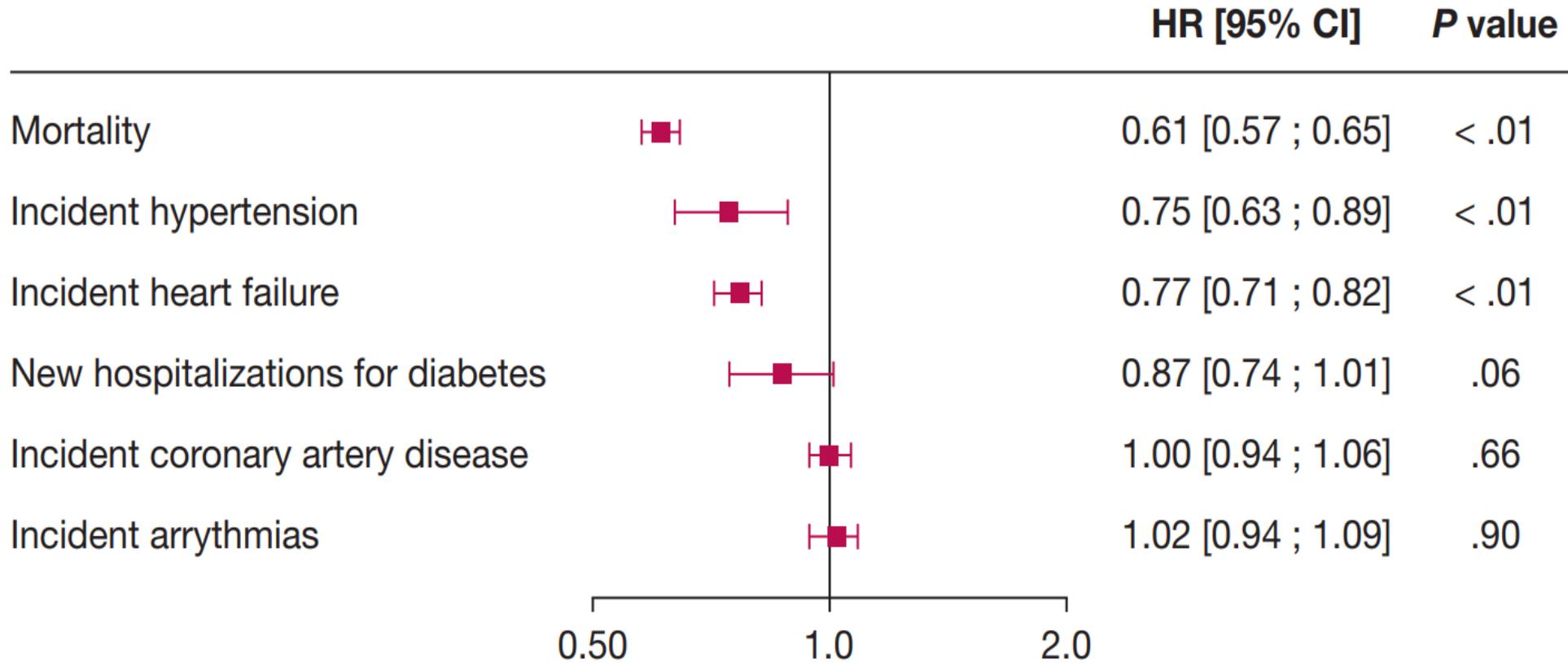
Probabilité de survie et de développer une insuffisance cardiaque après l'arrêt de la PPC



Relationship Between CPAP Termination and All-Cause Mortality

French Nationwide Database Analysis

Pépin et al Chest 2022



> Am J Respir Crit Care Med. 2022 Jul 11. doi: 10.1164/rccm.202202-0366OC. Online ahead of print.

Positive Airway Pressure Adherence, Mortality and Cardio-Vascular Events in Sleep Apnea Patients

Chloé Gervès-Pinquier¹, Sebastien Bailly², François Goupil³, Thierry Pigeanne⁴,
Sandrine Launois^{5,6}, Laurene Leclair-Visonneau⁷, Philippe Masson⁸, Acya Bizieux-Thaminy⁹,
Margaux Blanchard¹⁰, AbdelKebir Sabil¹¹, Dany Jaffuel¹², Jean-Louis Racineux¹³,
Wojciech Trzepizur¹⁴, Frédéric Gagnadoux¹⁵, Pays de la Loire Sleep Cohort Study Group

Pays de La Loire cohort: 5,138 patients, âge 64 (55-73)

Outcome: major adverse cardiovascular event (MACE)

Comparison de l'incidence des MACE selon l'adhérence à la PPC: <4H vs 4-6h, 6-7h and

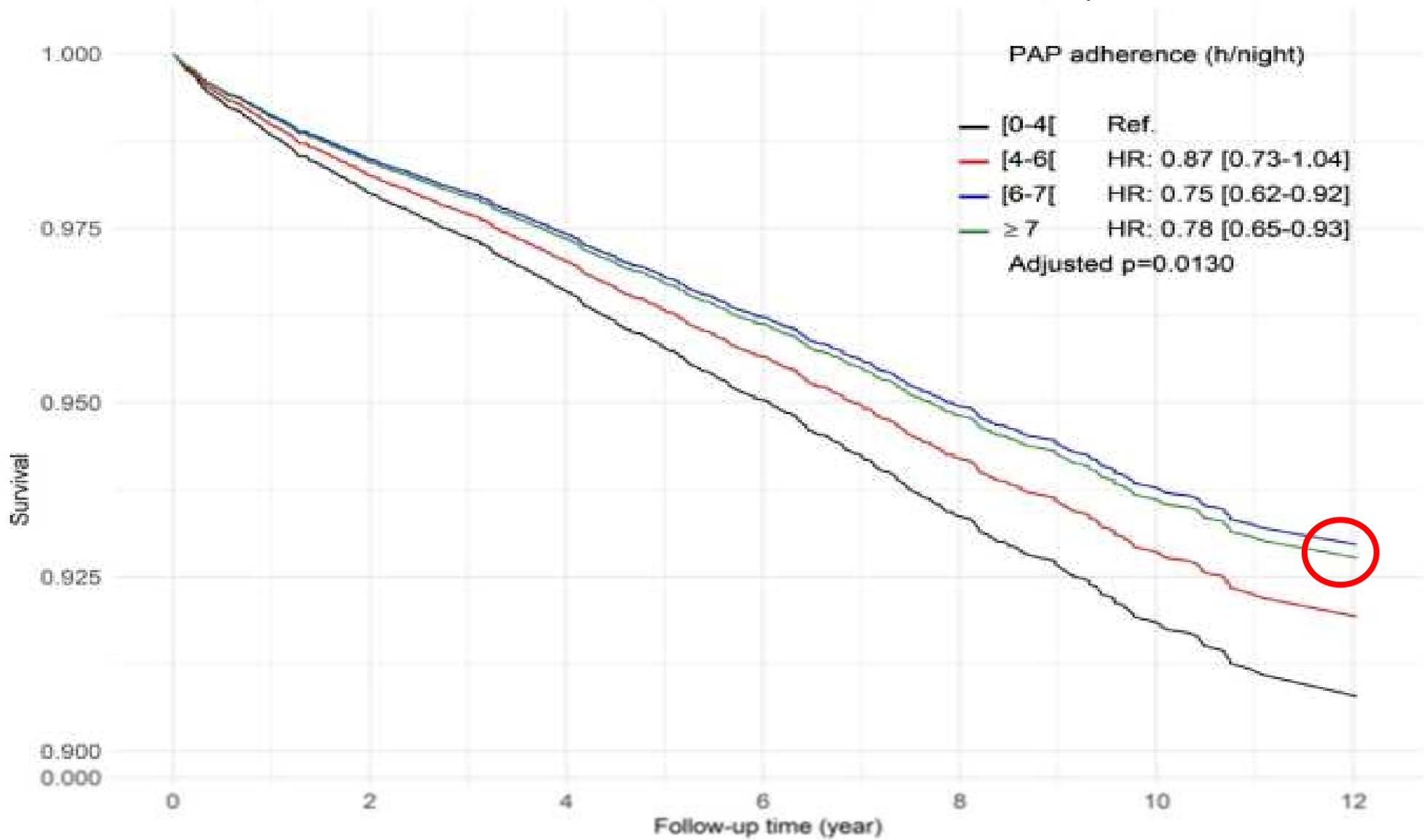
adjustment pour toutes les variables de risqué CV en utilisant le "inverse probability of

treatment weighting" (IPTW) le "medication possession ratio" pour limiter le "healthy u

as

Incidence des MACEs selon l'adhérence à la PPC

Gervès Pinquié AJRCCM 2022

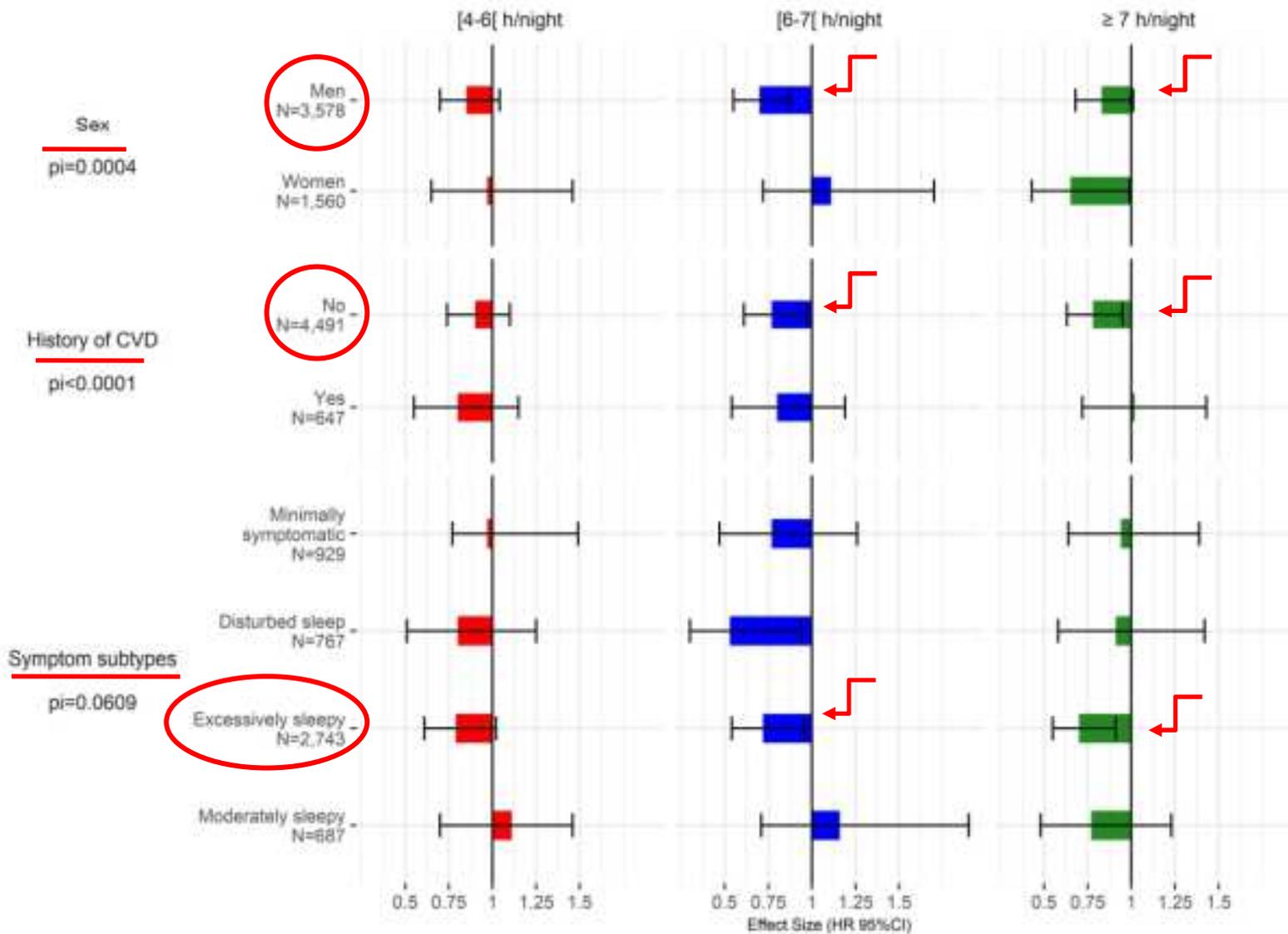


Analyses de sous-groupes selon sexe, mal CV et symptômes

Sex

Mal. CV

Symptômes



Prediction de l'effet de la PPC sur le risque CV avec des nouveaux marqueurs ?

- Apnea specific heart rate response (Δ HR)
- Hypoxic burden
- PWAD index

Hypoxic burden to guide CPAP treatment allocation in patients with obstructive sleep apnoea: a *post hoc* study of the ISAACC trial

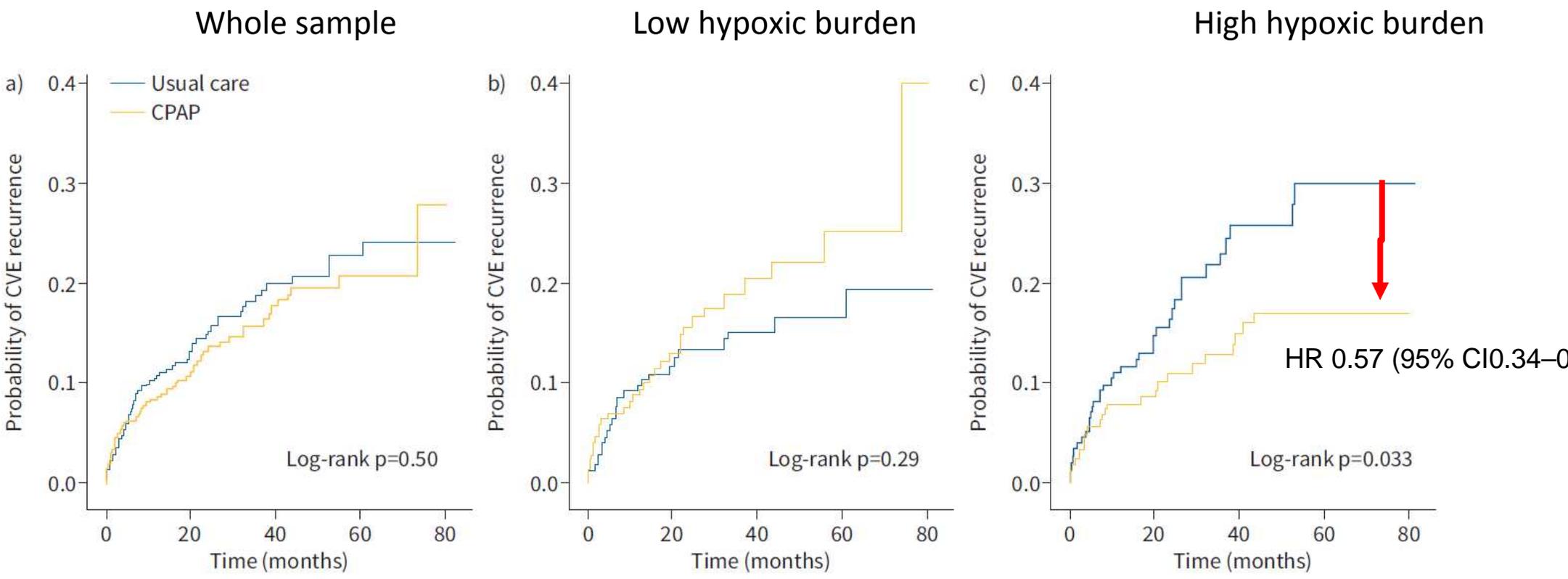
Lucía Pinilla ^{1,2,3,17}, Neda Esmaeili^{3,4,17}, Gonzalo Labarca ³, Miguel Ángel Martínez-García^{2,5}, Gerard Torres^{1,2}, Esther Gracia-Lavedan^{2,6}, Olga Mínguez⁶, Dolores Martínez⁶, Jorge Abad^{2,7}, Maria José Masdeu^{2,8}, Olga Mediano^{2,9}, Carmen Muñoz¹⁰, Valentín Cabriada ¹¹, Joaquín Duran-Cantolla^{2,12}, Mercè Mayos ^{2,13}, Ramón Coloma ¹⁴, Josep María Montserrat^{2,15}, Mónica de la Peña¹⁶, Wen-Hsin Hu³, Ludovico Messineo³, Mohammadreza Sehhati⁴, Andrew Wellman³, Susan Redline³, Scott Sands³, Ferran Barbé^{2,6}, Manuel Sánchez-de-la-Torre ^{1,2,18} and Ali Azarbarzin ^{3,18}

ERJ 20

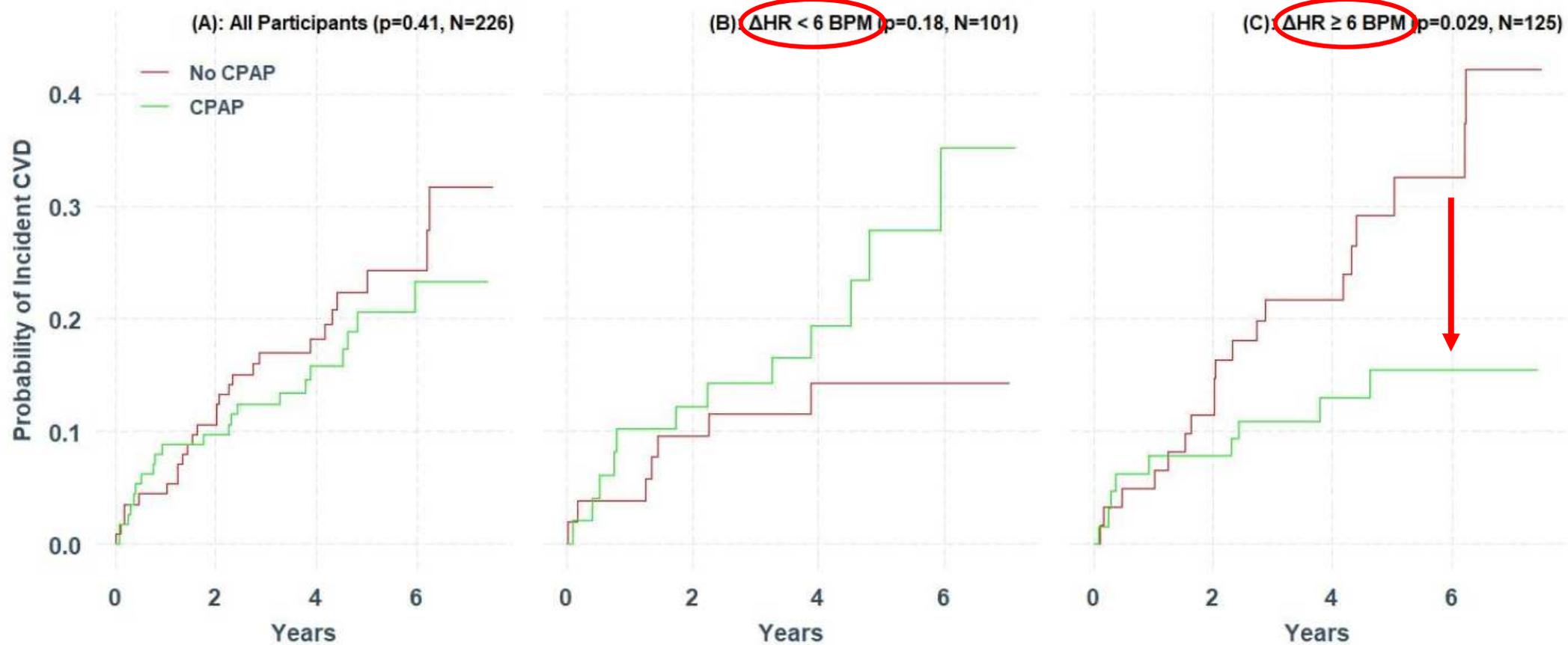
Low HB				
Usual care	26 (13.9)	33.34 (15.03–54.04)	Reference	
CPAP	31 (17.4)	24.34 (13.80–43.37)	1.33 (0.79–2.25)	0.281
High HB				
Usual care	36 (20.3)	24.26 (12.27–41.86)	Reference	
CPAP	24 (13.0)	32.00 (13.27–58.22)	0.57 (0.34–0.96)	0.035

A high Hypoxic burden is associated with protective effect of CPAP on incident cardiovascular events

Post hoc analysis of ISAACC trial (secondary prevention in non sleepy OSA)

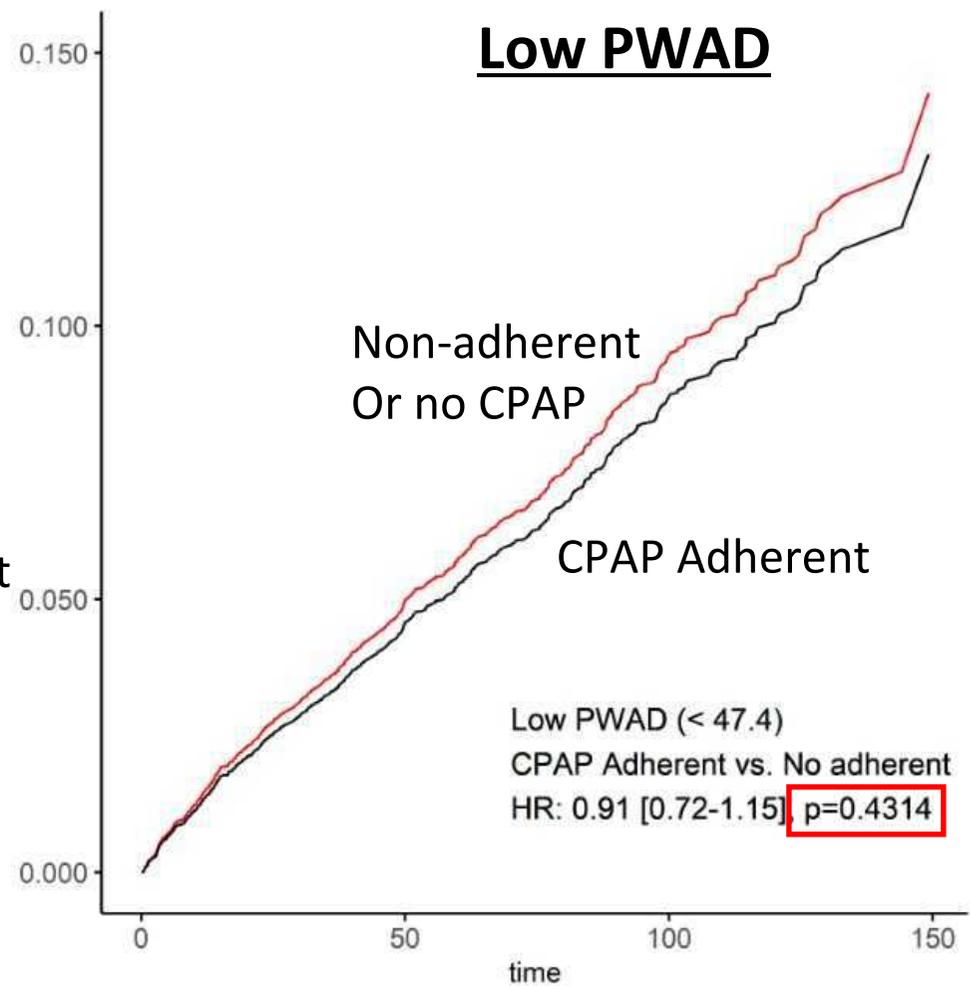
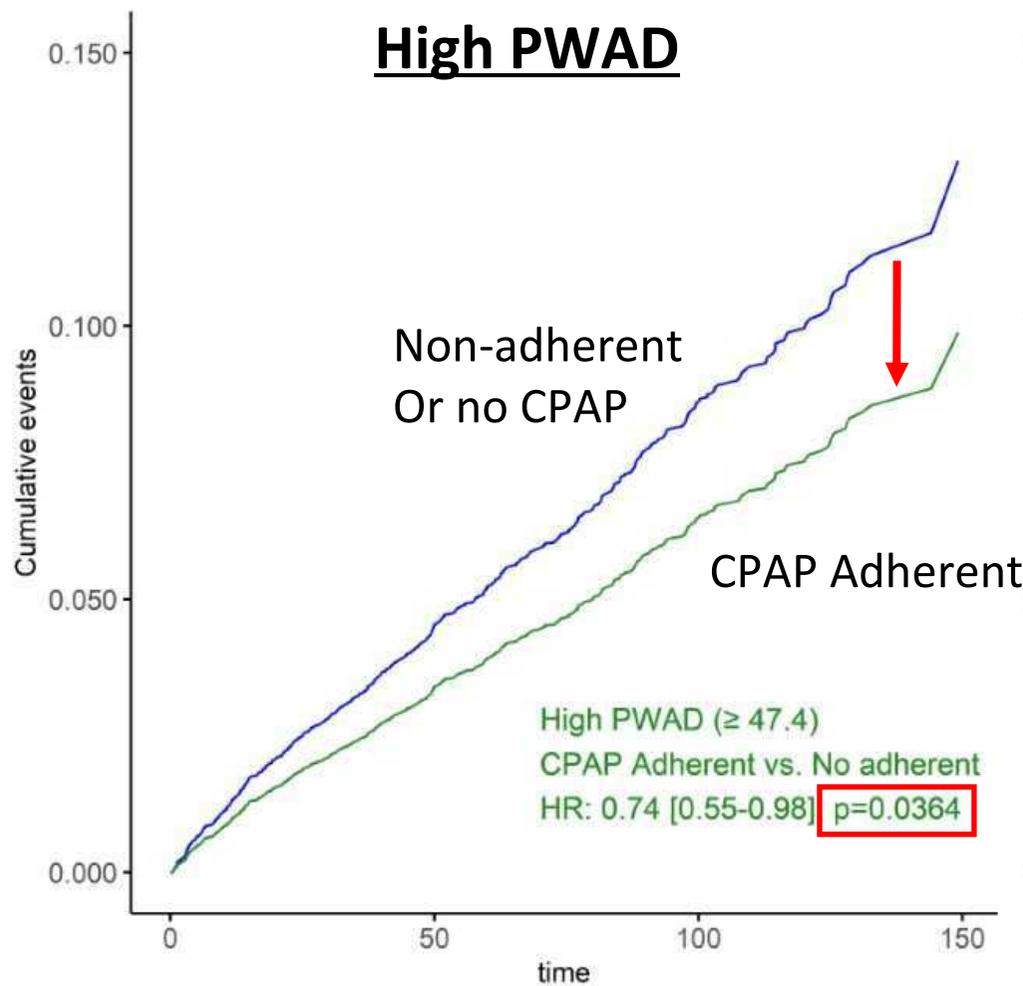


Patients with higher Δ HR exhibit greater cardiovascular benefit from CPAP therapy (RICCADSA study 2nd prevention in CV patients) Azarbazin A AJRCCM 20

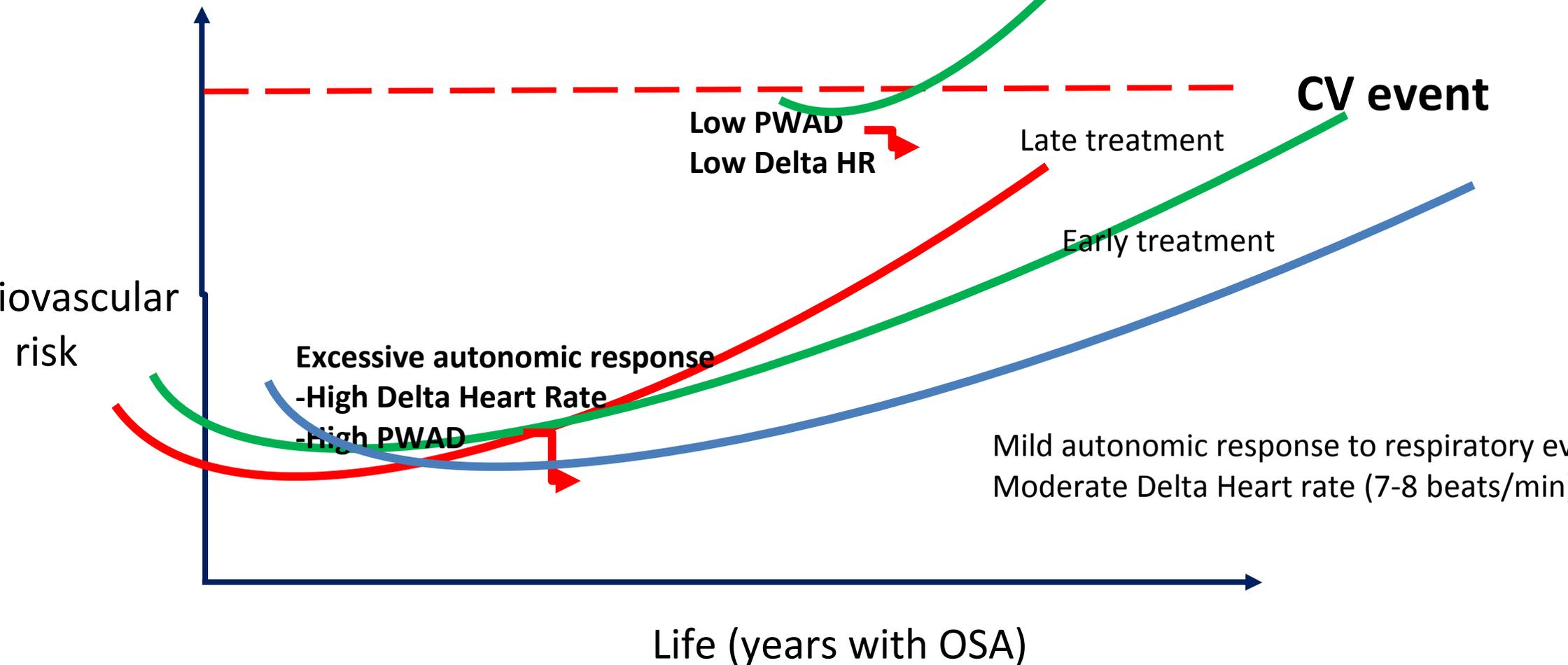


PAP adherence and CV risk according to PWAD index

SC cohort: Adjusted for Age, sex, BMI, diabetes, hypertension, tobacco, site, type of recorder (PG/PSG), Vasodilator



Hypothesis on the potential impact of CPAP on CV Risk in OSA



Conclusions

- . Dans les cohortes populationnelles, on trouve fréquemment un IAH élevé.
- . L'IAH augmente avec l'âge, l'obésité, le sexe masculin et la ménopause
- . L'IAH n'est pas un bon prédicteur du risque cardiovasculaire ou de la réponse au traitement par PPC en termes de prévention CV *secondaire*
- . D'autres marqueurs (evt combinés à l'IAH) pourraient permettre de mieux définir le risque cardiovasculaire dans l'OSAS
- . De nouvelles études interventionnelles avec PPC devraient être effectuée en se basant sur une meilleure sélection des patients OSAS à haut risque cardiovasculaire

Merci pour votre attention



Sleep apnoea in the asymptomatic elderly: a real issue for the brain?

Sébastien Celle^{1,2}, Claire Boutet ^{3,4}, Cédric Annweiler^{5,6},
Jean-Claude Barthélémy^{1,2} and Frédéric Roche^{1,2}

ERJ 2018

IRM cérébrale sur 226 participants asymptomatiques > 65 ans (age moyen 75.3±0.9) de la cohort PROOF (Evaluation of Ageing, Autonomic Nervous System Activity and Cardiovascular Events)

Résultats:

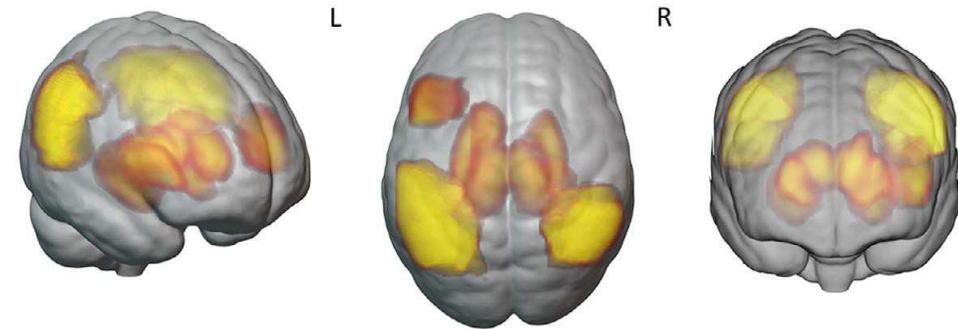
«L'IAH n'était associé à aucun changement en termes de volume cerebral, épaisseur du cortex ou surface du cortex cerebral»

Mean Oxygen Saturation during Sleep Is Related to Specific Brain Atrophy Pattern

Nicola Andrea Marchi, MD^{1,2}, Cristina Ramponi, PhD,¹ Camila Hirotsu, PhD,² José Haba-Rubio, MD,² Antoine Lutti, PhD,¹ Martin Preisig, MD,³ Pedro Marques-Vidal, MD, PhD,⁴ Peter Vollenweider, MD,⁴ Ferath Kherif, PhD,¹ Raphael Heinzer, MD, MPH,^{2†} and Bogdan Draganski, MD^{1,5†}

N=775 participants of the HypnoLaus study (Lausanne, CH)

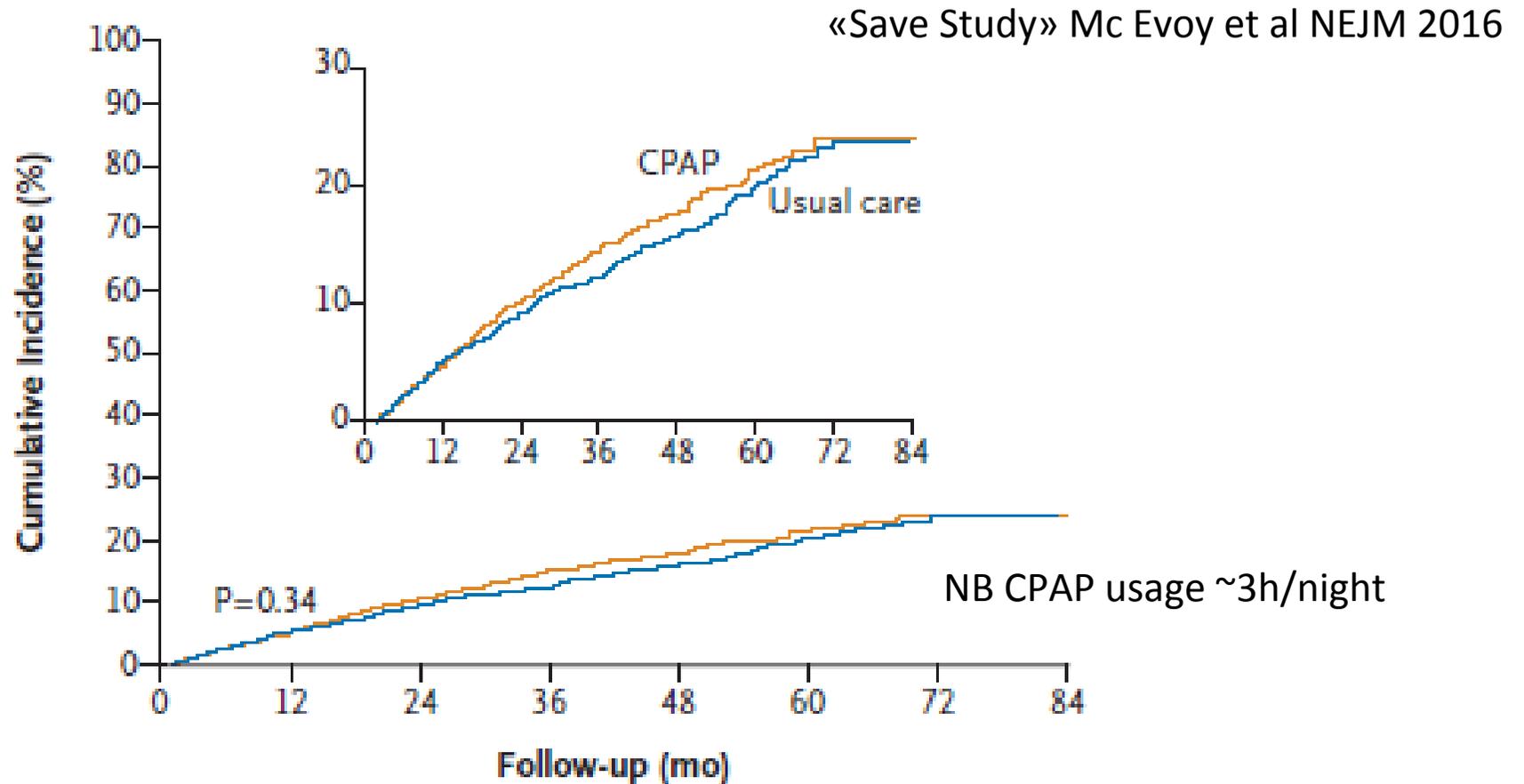
Age 60.3 y



Nocturnal hypoxemia is associated with grey matter atrophy, including in regions that are pivotal for cognition (e.g., hippocampus)

	Mean SaO2		Lowest SaO2		Log10(T90 + 1)		Log10(AHI + 1)		Log10(ODI + 1)	
	B	p	B	p	B	p	B	p	B	p
Total gray matter	1.097	0.031	0.015	0.915	-1.230	0.548	-0.499	0.802	-1.469	0.000
Subcortical structures										
R hippocampus	0.013	0.001 ^a	<0.001	0.910	-0.015	0.379	0.003	0.862	-0.008	0.000
L hippocampus	0.014	<0.001 ^a	<0.001	0.962	-0.013	0.403	<0.001	0.988	-0.009	0.000
R amygdala	0.003	0.036	0.001	0.773	-0.005	0.297	-0.003	0.489	-0.004	0.000
L amygdala	0.003	0.010 ^a	<0.001	0.988	-0.001	0.425	-0.005	0.320	-0.005	0.000
R thalamus	0.030	<0.001 ^a	-0.001	0.755	-0.053	0.093	0.034	0.264	<0.001	0.000
L thalamus	0.032	<0.001 ^a	<0.001	0.999	-0.056	0.086	0.014	0.661	-0.013	0.000
R caudate	0.014	0.014	<0.001	0.802	-0.008	0.713	0.001	0.955	0.001	0.000
L caudate	0.014	0.008 ^a	0.001	0.679	-0.012	0.579	0.005	0.796	0.004	0.000
R putamen	0.022	0.001 ^a	<0.001	0.436	-0.041	0.118	0.012	0.647	-0.002	0.000
L putamen	0.022	0.001 ^a	-0.002	0.276	-0.029	0.295	0.005	0.853	-0.006	0.000
R pallidum	0.004	0.043	<0.001	0.699	-0.013	0.141	0.008	0.340	0.006	0.000
L pallidum	0.002	0.307	<0.001	0.701	-0.009	0.319	0.008	0.364	0.008	0.000
Cortical structures										
R middle frontal gyrus	0.051	0.039	0.002	0.730	-0.126	0.210	-0.029	0.768	-0.102	0.000
L middle frontal gyrus	0.049	0.073	0.002	0.799	-0.086	0.436	-0.039	0.720	-0.123	0.000
R inferior frontal gyrus	0.026	0.035	<0.001	0.930	-0.097	0.053	-0.067	0.175	-0.077	0.000
L inferior frontal gyrus	0.038	0.004 ^a	0.007	0.051	-0.135	0.011	-0.066	0.196	-0.098	0.000
R superior temporal gyrus	0.020	0.057	-0.004	0.128	<0.001	0.998	0.033	0.421	0.001	0.000
L superior temporal gyrus	0.027	0.015	-0.002	0.420	-0.035	0.435	0.041	0.348	0.021	0.000
R middle temporal gyrus	0.035	0.055	<0.001	0.965	-0.020	0.783	0.010	0.884	-0.038	0.000
L middle temporal gyrus	0.038	0.038	-0.007	0.130	-0.014	0.847	0.047	0.518	0.009	0.000
R postcentral gyrus	0.037	0.020	0.002	0.690	-0.039	0.548	0.002	0.968	-0.056	0.000
L postcentral gyrus	0.010	0.556	-0.003	0.541	0.053	0.447	-0.025	0.709	-0.063	0.000
R supramarginal gyrus	0.022	0.082	-0.001	0.657	0.016	0.745	0.015	0.762	0.011	0.000
L supramarginal gyrus	0.041	0.003 ^a	0.004	0.329	-0.031	0.580	0.021	0.705	-0.019	0.000
R angular gyrus	0.039	0.011 ^a	0.001	0.769	-0.043	0.491	0.041	0.502	0.008	0.000
L angular gyrus	0.048	0.001 ^a	<0.001	0.905	-0.047	0.410	0.004	0.942	-0.008	0.000

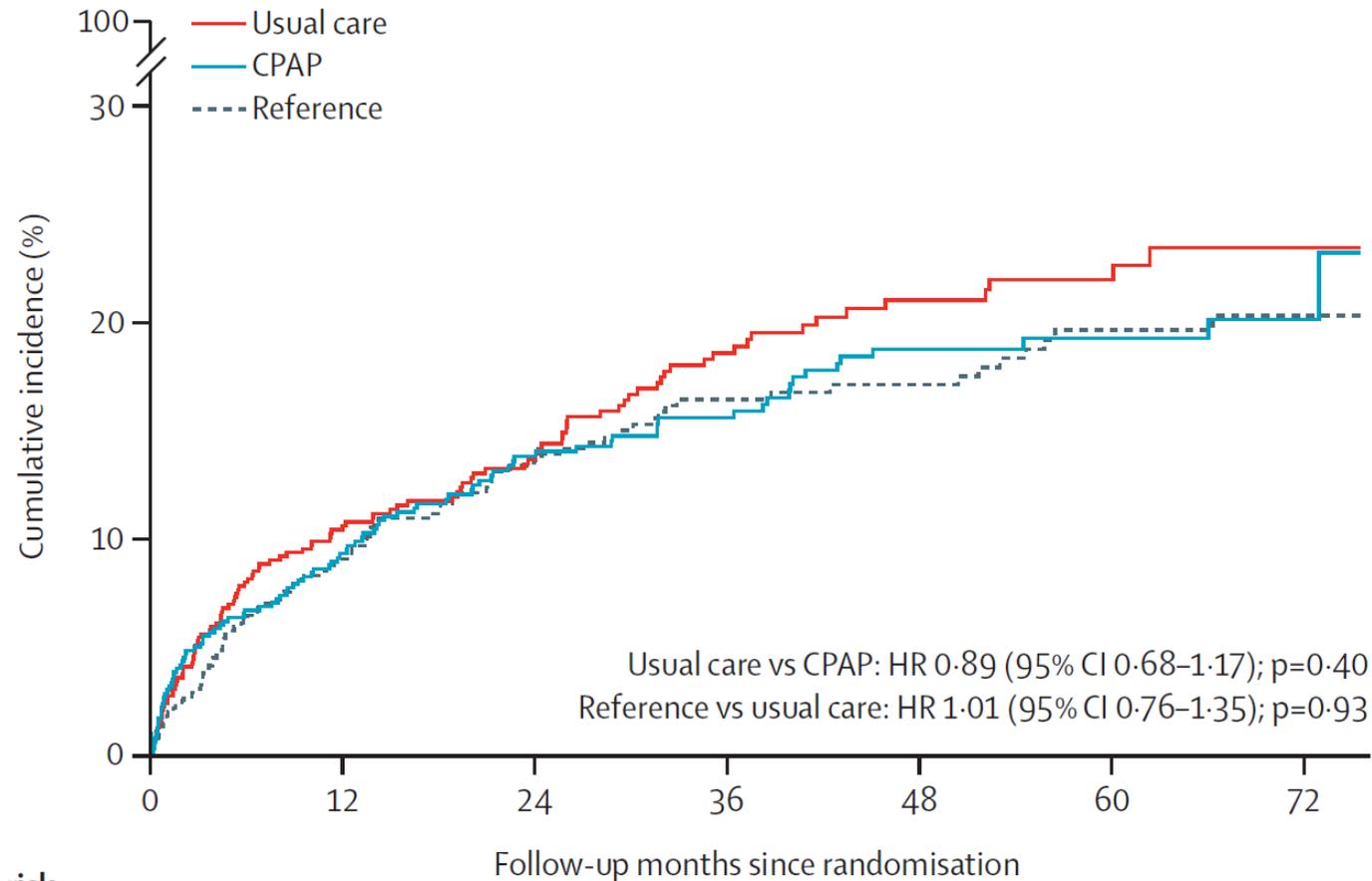
Sélection sur la base de l'IAH seul: pas de réponse détectable à la PPC en prévention secondaire



*Death from cardiovascular causes, myocardial infarction, stroke, or hospitalization for heart failure, unstable angina, or transient ischemic attack

Pas d'effet de la PPC en prévention secondaires si IAH >15/h: ISAACC Study

Sanchez-de-la Torre, Lancet RM 2019



Number at risk
(number censored)

Non-OSA	596 (0)	467 (78)	329 (197)	271 (245)	220 (294)	171 (337)	86 (422)
Usual care	626 (0)	498 (67)	376 (173)	276 (254)	185 (339)	126 (394)	46 (473)
CPAP	629 (0)	502 (74)	376 (176)	281 (263)	219 (315)	129 (404)	48 (484)

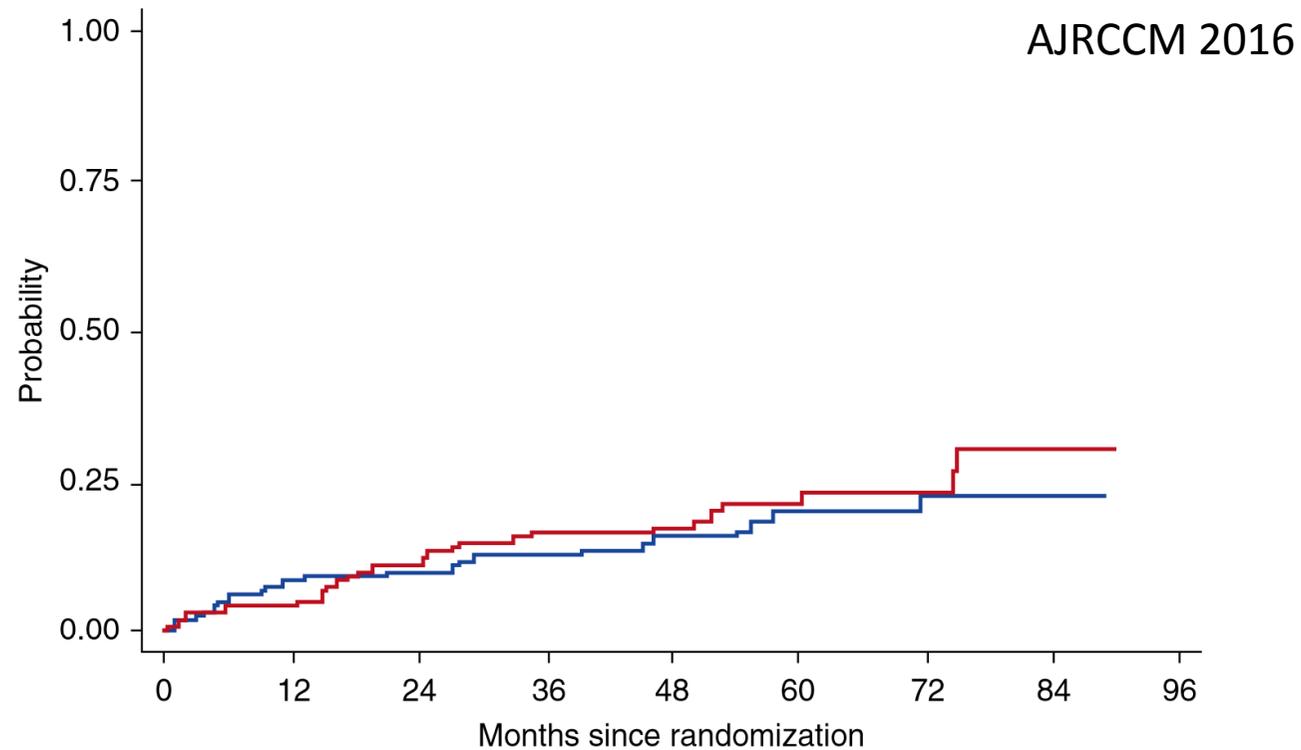
multicentrique
4 patients avec
nément CV préalable
vs TTT standard seul
férence AHI < 15/h

Effect of Positive Airway Pressure on Cardiovascular Outcomes in Coronary Artery Disease Patients with Nonsleepy Obstructive Sleep Apnea

The RICCADSA Randomized Controlled Trial

Yüksel Peker^{1,2}, Helena Glantz^{1,3}, Christine Eulenburg⁴, Karl Wegscheider⁴, Johan Herlitz^{5,6}, and Erik Thunström^{1,7}

Patients with coronary artery disease and obstructive sleep apnea (OSA) with an apnea-hypopnea index (AHI) >15/h and an oxygen desaturation index (ODI) >10/h. Randomized to CPAP (n=122) vs TTT (n=122)



Number at risk

CPAP	122	111	108	90	74	45	29	5	0
Control	122	117	109	87	71	43	27	5	0



Symptom Subtypes of Obstructive Sleep Apnea Predict Incidence of Cardiovascular Outcomes

Diego R. Mazzotti^{1,2*}, Brendan T. Keenan², Diane C. Lim^{1,2}, Daniel J. Gottlieb^{3,4,5},
Jinyoung Kim^{2,6}, Allan I. Pack^{1,2}

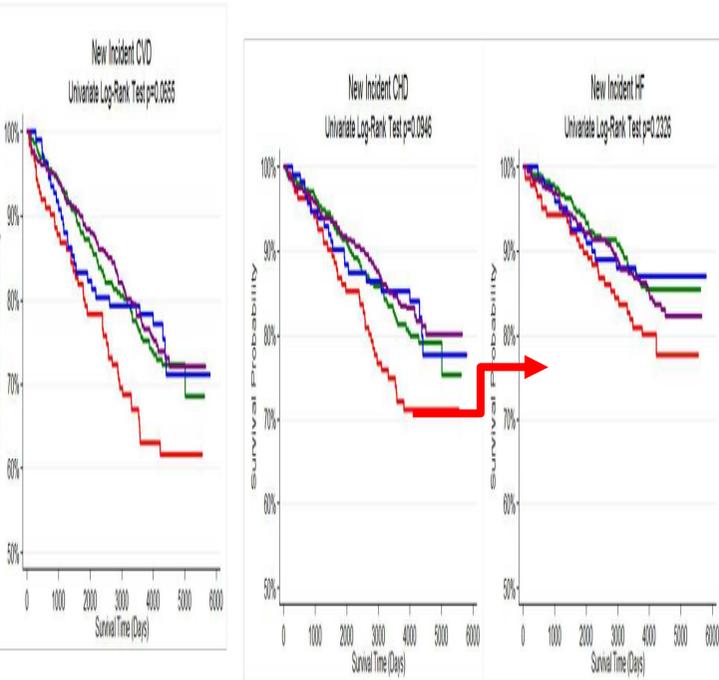
AJRCCM 2019

1,207 patients (apnea-hypopnea index [AHI] ≥ 15 /h) issus de la Sleep heart health study avec suivi prospectif des événements coronariens

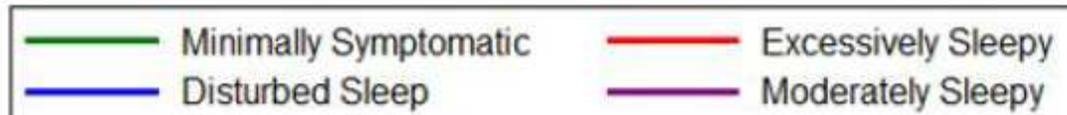
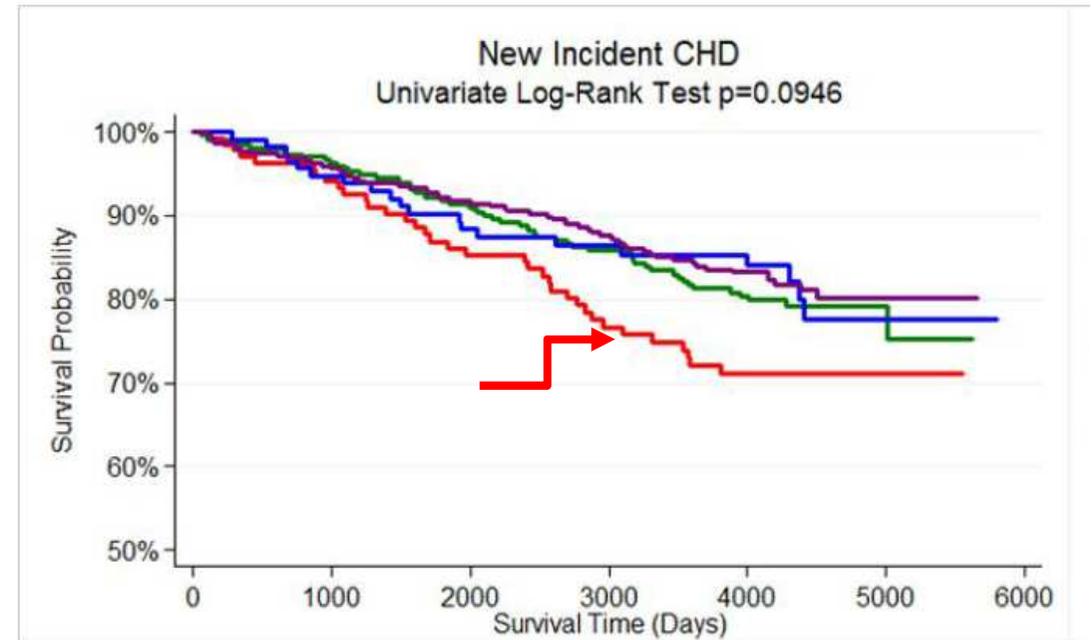
4 “clusters” de symptômes:

- *Disturbed Sleep* [12.2%]
- *Minimally Symptomatic* [32.6%]
- *Excessively Sleepy (mean ESS 13.7)* [16.7%]
- *Moderately Sleepy (mean ESS 10.6)* [38.5%]

Suivie sans maladie cardiovasculaire



Survie sans événement coronarien



Quel est l'IAH d'une population «normale»

Definition:

1. Absence de somnolence diurne (Epworth < 11)
2. IMC <25 kg/m² et prise de poids <15% en 5 ans
3. Absence d'hypertension artérielle
4. Absence de diabète
5. Absence de syndrome métabolique
6. Absence de maladie cardiovasculaire actuelle ou passée
7. Absence de dépression

IAH chez les «Super

N= 347 (123 hommes, 224 femmes)

healthy»

	Mean AHI	Median AHI	Range
Chicago 1999	8.1/h	4.7/h	0 – 66.1
AASM 2007	3.4/h	1.5/h	0 – 40.7
AASM 2013	7,4/h	4.1/h	0 – 63.4

Variables d'hypoxémie et incidence des maladies cardiovasculaires à 4.5 ans dans HypnoLaus

	Model 1		Model 2	
	HR (95% CI)	p-value	HR (95% CI)	p-value
% ODI, events/h	1.028 [1.007-1.049]	0.010	1.037 [0.985-1.040]	0.164
Mean SpO2, %	0.856 [0.749-0.978]	0.022	0.877 [0.761-1.010]	0.068
Time with Sao2 <90 %	1.014 [1.002-1.026]	0.019	1.013 [1.001-1.026]	0.034
Hypoxic burden, %min/h	1.007 [1.001-1.014]	0.023	1.005 [0.994-1.015]	0.376

Model 1: age, sex, body mass index, smoking, alcohol consumption, hyperlipidemia and diabetes

Model 2: additionally adjusted for apnea-hypopnea index and hypertension

Prevalence apnées du sommeil: Wisconsin 2011

Table 2. Model-based^a Prevalence Estimates of Mild to Severe Sleep-Disordered Breathing, Wisconsin Sleep Cohort Study, Wisconsin, 1988–2011

Body Mass Index ^b by Age, years	Estimated Prevalence of AHI ^c ≥5	
	% ^d	95% CI
<i>Men</i>		
50–70		
<25	18.9	14.8, 23.8
25–29.9	36.6	32.8, 40.3
30–39.9	61.4	57.0, 65.5
≥40	82.8	77.1, 87.7
<i>Women</i>		
50–70		
<25	9.3	6.8, 12.3
25–29.9	20.2	16.4, 24.4
30–39.9	41.1	35.6, 46.7
≥40	67.9	60.6, 75.1

Prevalence OSAS: Wisconsin 2013

Table 4. Model-based^a Prevalence Estimates of Mild to Severe Sleep-Disordered Breathing With Concomitant Daytime Sleepiness Wisconsin Sleep Cohort Study, Wisconsin, 1993–2011

Body Mass Index ^b by Age, years	Estimated Prevalence of <u>AHI^c >5 and ESS Score >10</u>	
	% ^d	95% CI
<i>Men</i>		
50–70		
<25	7.7	4.9, 10.9
25–29.9	13.8	11.0, 16.1
30–39.9	24.9	20.7, 29.4
≥40	43.0	33.8, 53.9
<i>Women</i>		
50–70		
<25	2.8	1.6, 4.4
25–29.9	5.3	3.8, 7.1
30–39.9	10.5	7.8, 13.2
≥40	20.9	15.4, 27.8