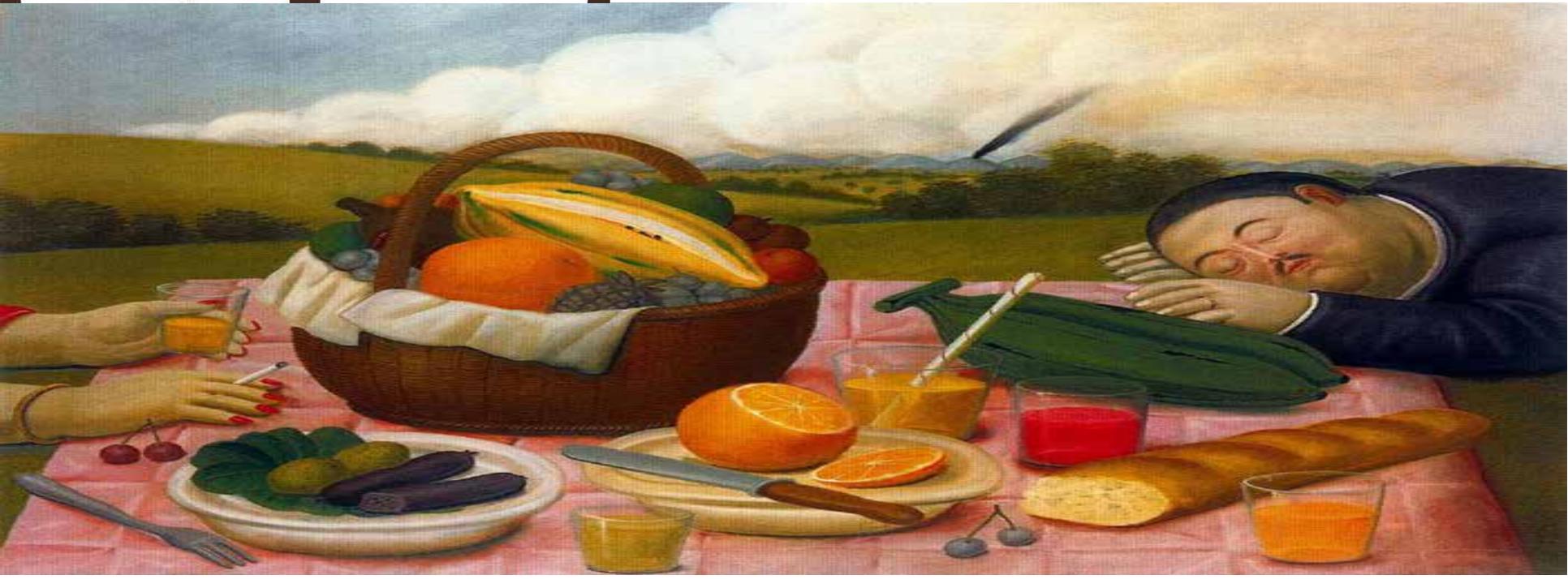


Dr. Jorge Abad
Servicio Neumología H.GTiP

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15 i 16 d'abril 2016

ALTERACIONES METABOLICAS Y SAHS



HOMBRES



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ataque
de

NERVIOS

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CIRCULATING FABP4 AND FABP5 LEVELS IN OSA SEVERITY AND TREATMENT

<http://dx.doi.org/10.5665/sleep.3210>

Circulating FABP4 and FABP5 Levels Are Differently Linked to OSA Severity and Treatment

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Impact of OSA on Biological Markers in Morbid Obesity and Metabolic Syndrome

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Background and Objective: There is compelling evidence that obstructive sleep apnoea (OSA) can affect metabolic syndrome (MetS) and cardiovascular risk, but the intermediate mechanisms through which it occurs have not been well defined. We explored the impact of OSA in morbidly obese patients with MetS on adipokines, pro-inflammatory markers, endothelial dysfunction, and atherosclerosis markers.

Methods: We included 52 morbidly obese patients in an observational study matched for age, gender and central obesity in 3 groups (OSA-MetS, Non-OSA-MetS, and Non OSA-non-MetS). Anthropometrical, blood pressure, and fasting blood measurements were obtained the morning after an overnight polysomnography. VEGF, soluble CD40 ligand (sCD40L), TNF- α , IL-6, leptin, adiponectin, and chemerin were determined in serum by ELISA. OSA was defined as apnea/hypopnea index ≥ 15 and MetS by NCEP-ATP III.

Results: Cases and control subjects did not differ in age, BMI, waist circumference, and gender (43 ± 10 years, 46 ± 5 kg/m², 128 ± 10 cm, 71% females). The cases had severe OSA with

47 (32-66) events/h, time spent $< 90\%$ SpO₂ 7% (5%-31%). All groups presented similar serum cytokines, adipokines, VEGF, and sCD40L levels.

Conclusions: In a morbidly obese population with established MetS, the presence of OSA did not determine any differences in the studied mediators when matched by central obesity. Morbidly obese NonOSA-NonMetS had a similar inflammatory, adipokine VEGF, and sCD40L profile as those with established MetS, with or without OSA. Obesity itself could overwhelm the effect of sleep apnea and MetS in the studied biomarkers.

Keywords: Obstructive sleep apnoea, obesity, metabolic syndrome, adipokines, inflammatory markers, endothelial dysfunction

Citation: Salord N; Gasa M; Mayos M; Fortuna-Gutierrez AM; Montserrat JM; Sánchez-de-la-Torre M; Barceló A; Barbé F; Vilarrasa N; Monasterio C. Impact of OSA on biological markers in morbid obesity and metabolic syndrome. *J Clin Sleep Med* 2014;10(3):263-270.

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Abbreviations

AHI	apnea–hypopnea index	MI	myocardial infarction
AMI	acute myocardial infarction	mPTP	mitochondrial permeability transition pore
AP1	activator protein1	NAC	N-acetylcysteine
BH ₄	tetrahydrobiopterin	NADPH	reduced nicotinamide adenine dinucleotide phosphate
CAD	coronary artery disease	nCPAP	nasal continuous positive airway pressure
CD	cluster of differentiation	NFκB	nuclear factor κB
cGMP	cyclic GMP	nNOS	neuronal NOS (NOS1)
CNS	central nervous system	NO	nitric oxide
COPD	chronic obstructive pulmonary disease	NOS	nitric oxide synthase
eNOS	endothelial NOS (NOS3)	Nox	NADPH oxidase
EPCs	endothelial progenitor cells	Nrf2	nuclear factor (erythroid-derived 2)-like2
EPO	erythropoietin	O ₂ ^{•-}	superoxide anion
Erk1/2	extracellular signal-regulated kinase	ODI	oxygen desaturation index
GPx	glutathione peroxidase	OH [•]	hydroxyl radical
GSH	glutathione (reduced)	OONO ⁻	peroxynitrite
GSSG	glutathione disulfide (oxidized)	OSA	obstructive sleep apnea
H ₂ O ₂	hydrogen peroxide	oxLDL	oxidized LDL
HDL	high-density lipoprotein	p38 MAPK	p38 MAP kinase
HIF-1α	hypoxia inducible factor-1α	PAC-1	specific marker for glycoprotein (GP)IIb/IIIa
HNA	4-hydroxy-2-nonenal	PI3K	phosphatidylinositol-3-kinase
HO-1	heme oxygenase 1	PKC	protein kinase C
HSPs	heat shock proteins	PON-1	paraxonase-1
ICAM-1	intracellular cell adhesion molecule 1	PSLG-1	P-selectin glycoprotein ligand 1
I/R	ischemia and reperfusion	Redox	oxidation/reduction balance
IH	intermittent hypoxia	RNS	reactive nitrogen species
IL-8	interleukin 8	ROS	reactive oxygen species
IL-6	interleukin 6	SDB	sleep disordered breathing
iNOS	inducible NOS (NOS2)	SH	thiol
IPC	ischemic preconditioning	SOD	superoxide dismutase
Keap1	Kelch-like ECH-associated protein 1	TBARS	thiobarbituric acid reactive substances
LAD	left anterior descending artery	TNF-α	tumor necrosis factor α
LDL	low-density lipoprotein	VCAM-1	vascular cell adhesion molecule 1
MDA	malonaldehyde	VEGF	vascular endothelial growth factor
		VEGF-R2 (KDR)	VEGF receptor 2

Oxidative stress in obstructive sleep apnea and intermittent hypoxia – Revisited – The bad ugly and good: Implications to the heart and brain



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Coronary collaterals

SUMMARY

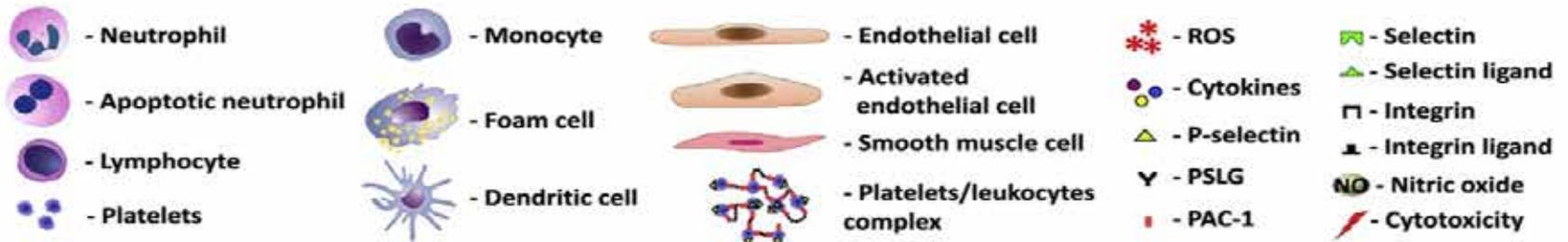
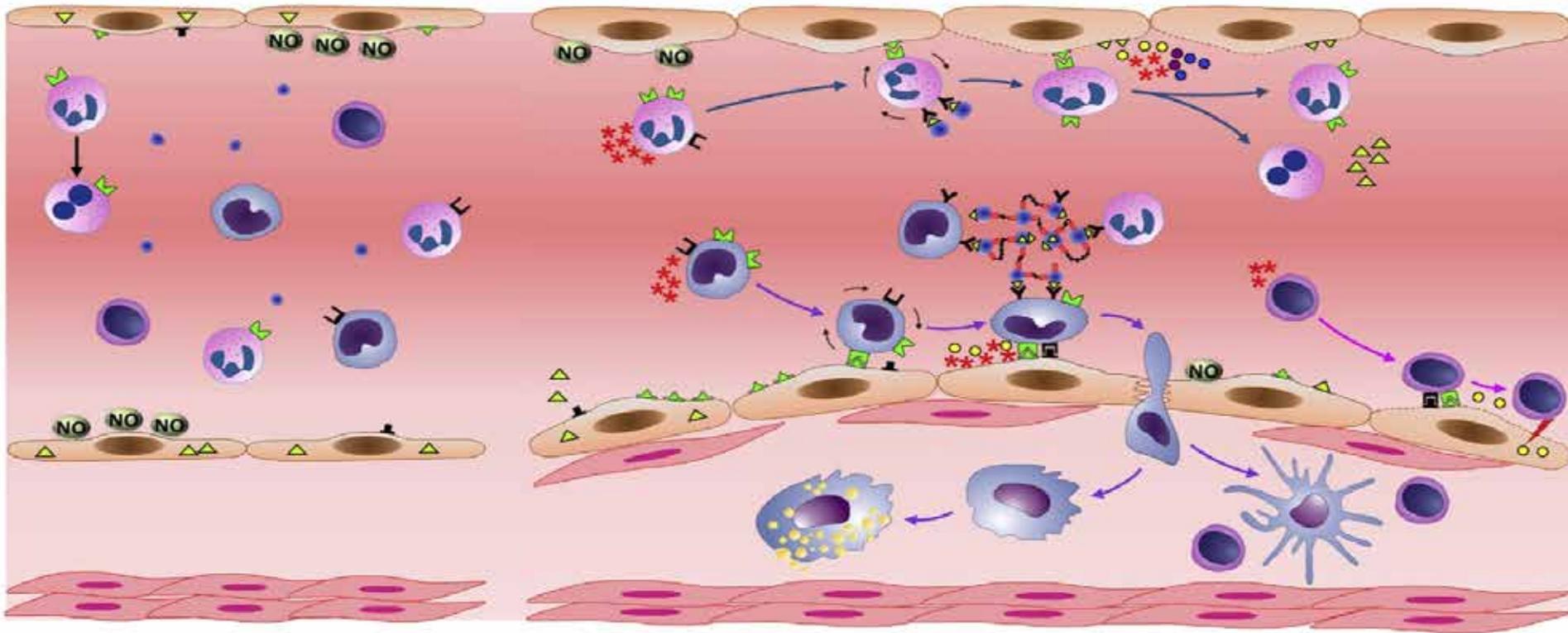
Obstructive sleep apnea (OSA), characterized by intermittent hypoxia (IH), is linked with increased reactive oxygen species/reactive nitrogen species (ROS/RNS) and oxidative stress, which adversely affect the associated cardio-/cerebro-vascular disease in OSA. Yet, animal and a small number of human studies support activation of cardio-/cerebro-protective mechanisms as well. ROS/RNS are intricate and multifaceted molecules with multiple functions. At low-moderate concentrations ROS/RNS are considered “good”, by regulating vital cellular functions. At higher levels, they are considered “bad” by promoting oxidative stress and damaging vital macromolecules through ischemia and reperfusion (I/R) injury. Subsequently, ROS/RNS can get “ugly” by eliciting sterile inflammation and a multitude of deadly pathologies. What makes ROS/RNS good, bad, or ugly? A dynamic interplay between a large number of factors determines the outcomes. These include the types of ROS/RNS produced, their quantity, duration, frequency, intracellular localization, micro-environmental antioxidants, as well as the genetic make-up and life style related variables. This review presents the currently available data on redox biology in physiological/pathophysiological conditions and in OSA/IH, in order to better understand the apparently contradictory findings on damage vs. repair. These findings are discussed within the context of the prevailing views on I/R associated ROS/RNS, and their potential implications to OSA.

- Cada vez hay mas evidencia que los trastornos del sueño , tienen implicaciones en las alteraciones metabólicas.
- No conocemos los efectos de los relojes biológicos (ritmos circadianos) en la regulación del metabolismo

- Las alteraciones metabólicas que provocan el SAHS y cualquier patología del sueño son confusas y en ocasiones contradictorias
- Hay interrelaciones complejas, que por diferentes mecanismos actúan sobre el metabolismo.

Normoxia

OSA/IH



Los leucocitos interactúan con las cel. endoteliales en IH. Los neutro, monocitos, linfos, plaqueta etc. Se activan con la HI produciendo mayores cantidades de ROS, moléculas de adhesión, citoquinas y disminución de NO promoviendo daño endotelial. La IH produce Cel. espumosa y dendríticas induciendo aún más la aterosclerosis.

MECANISMOS PATOGENICOS



FRAGMENTACION DEL SUEÑO



Sensibilidad insulina y
efectividad de la
glucosa



HIPOXIA INTERMITENTE

Efecto Lipolítico,
Apoptosis Cel. Beta,
Inhibe Glucogénesis



ACTIVIDAD SIMPÁTICA

ESTRÉS OXIDATIVO

↑ ROS, Colesterol, resistencia insulina

Pensamientos acelerados

Falta de memoria

Cansancio constante

Trastorno del sueño

Mayor negatividad

Irritabilidad

Incapacidad para disfrutar

Opresión en el pecho

Insomnio

Suspiros o bostezos frecuentes

Molestias digestivas

Sudoración excesiva

Estreñimiento o diarrea





Elevación
citoquinas ,Il6
Factor necrosis
tumoral alfa

INFLAMACION SISTEMICA

Grelina :adiposidad
Leptina :elevada obesidad
Resistina y Adiponectina



ALTERACION HORMONAL

OBESIDAD



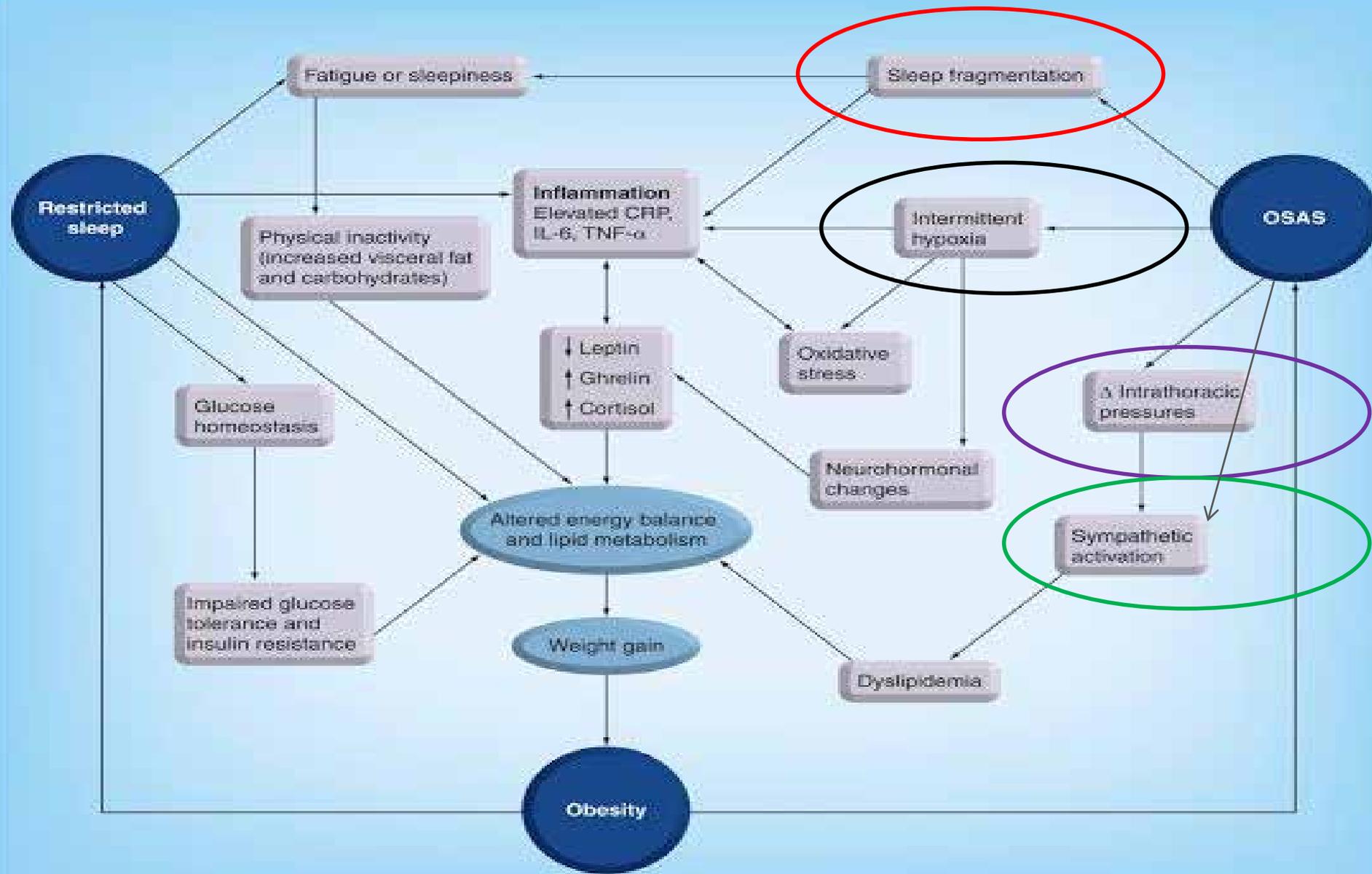


Figure 1. The circular model of sleep restriction, obstructive sleep apnea syndrome and obesity, emphasizing the central role of disrupted lipid metabolism

CRP: C-reactive protein; OSAS: Obstructive sleep apnea syndrome.

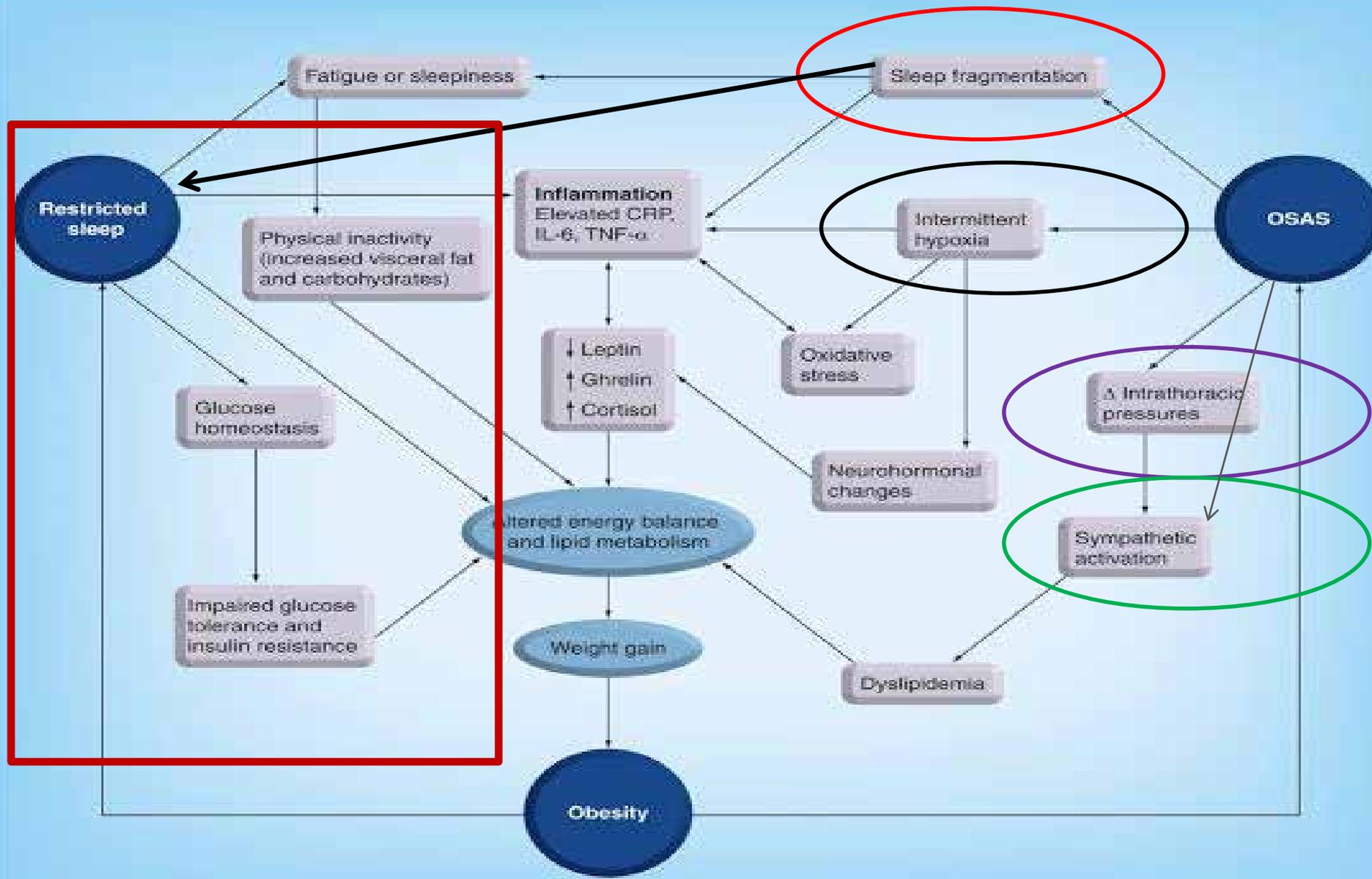


Figure 1. The circular model of sleep restriction, obstructive sleep apnea syndrome and obesity, emphasizing the central role of disrupted lipid metabolism

CRP: C-reactive protein; OSAS: Obstructive sleep apnea syndrome.



Durante los últimos 50 años la duración del Sueño en adultos y adolescentes ha disminuido de 1.5- 2 horas por noche en mas de un 30 % de la población americana



FRAGMENTACION DEL SUEÑO

- Impacto negativo sobre el metabolismo de la glucosa con:
 - Disminución la sensibilidad a la insulina y de la eficacia a la glucosa ,Inhibición de la secreción de insulina .
 - Aumento de la glucogénesis hepática
- Híperreactividad del eje HPA:
 - Aumento de los corticoides
 - Aumento de la grelina y disminución de la leptina,
 - Aumento de la ingesta de diurnas .
- Mayor estrés oxidativo y disminución e la capacidad antioxidante

SLEEP DURATION AND CARDIOMETABOLIC HEALTH

Contribution of Inflammation, Oxidative Stress, and Antioxidants to the Relationship between Sleep Duration and Cardiometabolic Health

Thirumagal Kanagasabai, MSc; Chris I. Arden, PhD

School of Kinesiology and Health Science, York University, Toronto, Ontario, Canada

Objectives: To explore the interrelationship and mediating effect of factors that are beneficial (i.e., antioxidants) and harmful (i.e., inflammation and oxidative stress) to the relationship between sleep and cardiometabolic health.

Design: Cross-sectional data from the 2005–2006 National Health and Nutrition Examination Survey.

Setting: Nationally representative population sample from the US.

Participants: Age ≥ 20 y with sleep data; final analytical sample of $n = 2,079$.

Interventions: N/A.

Measurements and Results: Metabolic syndrome was classified according to the Joint Interim Statement, and sleep duration was categorized as very short, short, adequate, and long sleepers (≤ 4 , 5–6, 7–8, and ≥ 9 h per night, respectively). The indirect mediation effect was quantified as large (≥ 0.25), moderate (≥ 0.09), modest (≥ 0.01), and weak (< 0.01). In general, inflammation was above the current clinical reference range across all sleep duration categories, whereas oxidative stress was elevated among short and very short sleepers. Select sleep duration–cardiometabolic health relationships were mediated by C-reactive protein (CRP), γ -glutamyl transferase (GGT), carotenoids, uric acid, and vitamins C and D, and were moderated by sex. Specifically, moderate-to-large indirect mediation by GGT, carotenoids, uric acid, and vitamin D were found for sleep duration–waist circumference and –systolic blood pressure relationships, whereas vitamin C was a moderate mediator of the sleep duration–diastolic blood pressure relationship.

Conclusions: Several factors related to inflammation, oxidative stress, and antioxidant status were found to lie on the casual pathway of the sleep duration–cardiometabolic health relationship. Further longitudinal studies are needed to confirm our results.

Keywords: antioxidants, cardiometabolic health, indirect mediation effect, inflammation and oxidative stress, sleep duration

Citation: Kanagasabai T, Arden CI. Contribution of inflammation, oxidative stress, and antioxidants to the relationship between sleep duration and cardiometabolic health. *SLEEP* 2015;38(12):1905–1912.

Analizan en 2.076 sujetos ,la relación entre duración de sueño con la inflamación / estrés oxidativo y las capacidades antioxidantes

- Las personas que dormían 7 horas ,tenían niveles de inflamación optima ,con un equilibrio entre el estrés oxidativo y los mecanismos antioxidantes .
- Una corta duración de sueño se asociaba a un aumento de PCR, GGT, Ac úrico y vit. A y niveles bajos de vit. C ,D, E .

Review Article

Sleep Deprivation and Oxidative Stress in Animal Models: A Systematic Review

**Gabriel Villafuerte,¹ Adán Miguel-Puga,¹ Eric Murillo Rodríguez,²
Sergio Machado,³ Elias Manjarrez,⁴ and Oscar Arias-Carrión¹**

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Ø ¿Hay diferencia en las propiedades antioxidantes entre sueño paradójico y sueño profundo?

- El sueño profundo : baja actividad metabólica y menor consumo de O₂.
- El sueño paradójico : se asocia a una elevada actividad metabólica y mayor consumo de O₂.
- El sueño paradójico juega un papel importante como elemento antioxidante.
- Su disminución aumenta el estrés oxidativo.

Ø *¿Que regiones son preferiblemente afectadas por el estrés oxidativo después de la deprivación de sueño?*

- El cerebro no se ve afectado de forma uniforme por la privación de sueño.
- Hay una peroxidación lipídica en el hipocampo ,tálamo e hipotálamo .Pero disminución de la peroxidación en la corteza

Ø El papel antioxidante del sueño ¿Se observa solo en el cerebro? .

- El sueño , regula los genes responsables de la síntesis de una serie de proteínas antioxidantes.
- El sueño cambia la actividad metabólica.
- Durante el sueño hay una disminución de la actividad metabólica ,reduciéndose la carga oxidativa y por lo tanto un beneficio para para el cerebro, corazón

Ø ¿El estrés oxidativo induce al sueño?

- El nivel de estrés oxidativo puede inducir el sueño.
- Hay una serie de moléculas endógenas que se acumulan en vigilia y esto podría activar un mecanismo antioxidante.

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Review article

Interactions between sleep, stress, and metabolism: From physiological to pathological conditions



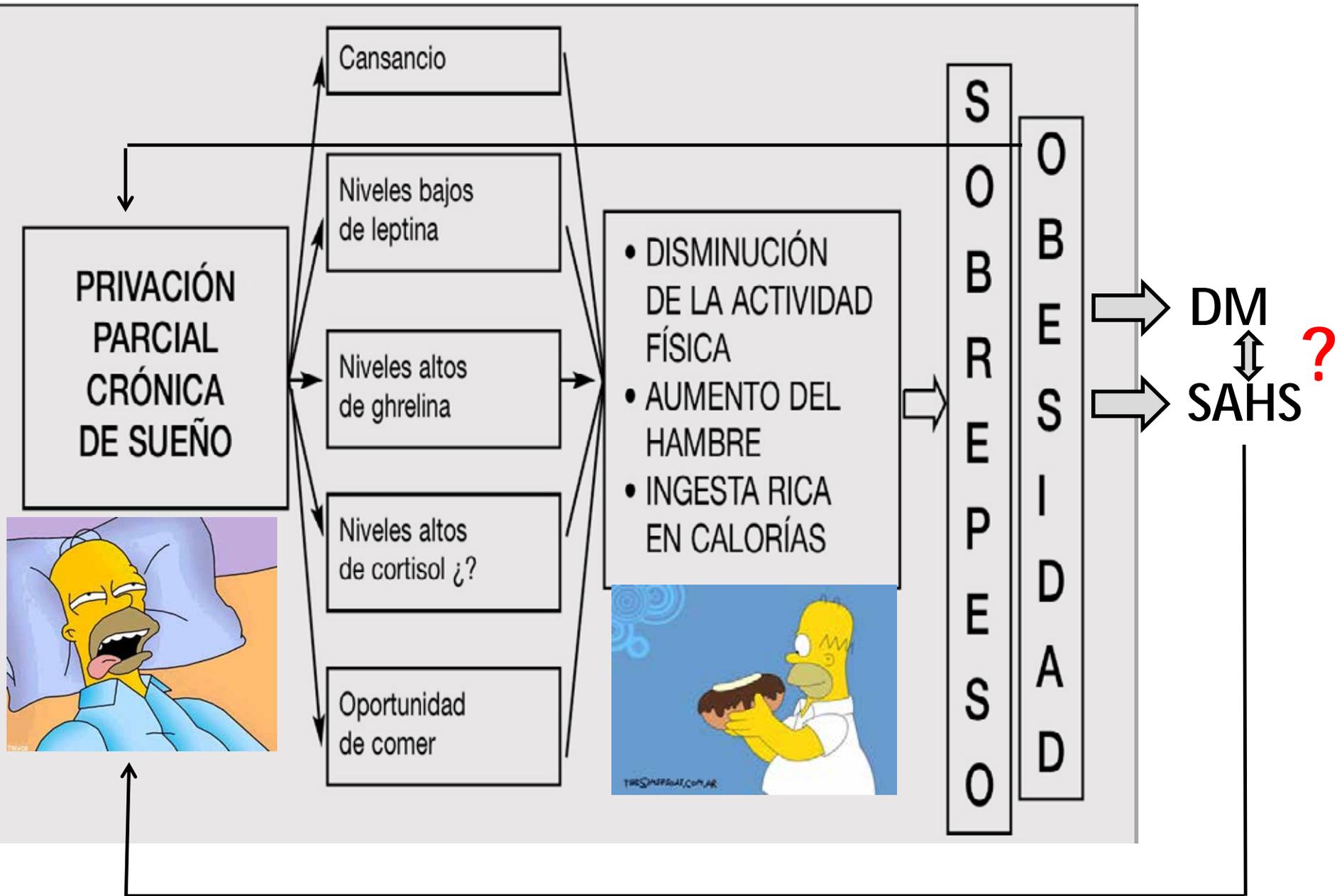
*Camila Hirotsu**, Sergio Tufik, Monica Levy Andersen

Department of Psychobiology, Universidade Federal de São Paulo, São Paulo, Brazil

- Dormir solo 4 horas durante 6 noches consecutivas en hombres jóvenes , mostró un aumento de los niveles de cortisol .
- La falta de sueño se relaciona con alteración del eje HPA.  Con efectos nocivos sobre el cuerpo .
- Sueño corto  mayor riesgo de obesidad y diabetes

- Reducción significativa de la insulina con una semana de restricción de sueño.
- La reducción del sueño produce una disminución de la leptina (18%) y aumento de la grelina (28%) que modifica la ingesta de los alimentos.







- Marshall en 2009 proporciono la primera evidencia de que el SAHS moderado es un factor de riesgo independiente para desarrollar diabetes . Después de un seguimiento durante 4 años , el 20% de los pacientes SAHS moderado / grave habían sido diagnosticados de DM.

ORIGINAL ARTICLE

**Obstructive sleep apnoea and the risk of type 2 diabetes:
A meta-analysis of prospective cohort studies**

XIA WANG,¹ YANPING BI,³ QIAN ZHANG² AND FANG PAN²

¹Department of Maternal and Child Health Care, School of Public Health and ²Institute of Medical Psychology, School of Medicine, Shandong University and ³Department of Emergency, Shandong Qianfoshan Hospital, Jinan, China

ABSTRACT

Background and objective: There has been increasing recognition that obstructive sleep apnoea (OSA) is associated with incident type 2 diabetes. The aim of this study was to assess the association between the severity of OSA and the risk of type 2 diabetes by performing a meta-analysis of all available prospective cohort studies.

SUMMARY AT A GLANCE

A meta-analysis of all eligible prospective cohort studies showed that moderate-severe obstructive sleep apnoea was associated with a greater risk of developing type 2 diabetes.

- Este metaanálisis incluían 5.953 pacientes con un seguimiento de 2,7-16 años
- El SAHS moderado grave se asocia con un mayor riesgo de DM cuando se compara con los no SAHS .
- Los SAHS leves no mostraron un riesgo superior a los no SAHS.

REVIEW

Open Access

The impact of sleep disorders on glucose metabolism: endocrine and molecular mechanisms

Anne Briançon-Marjollet^{1,2}, Martin Weiszenstein³, Marion Henri^{1,2}, Amandine Thomas^{1,2},
Diane Godin-Ribuot^{1,2†} and Jan Polak^{3,4,5*†}

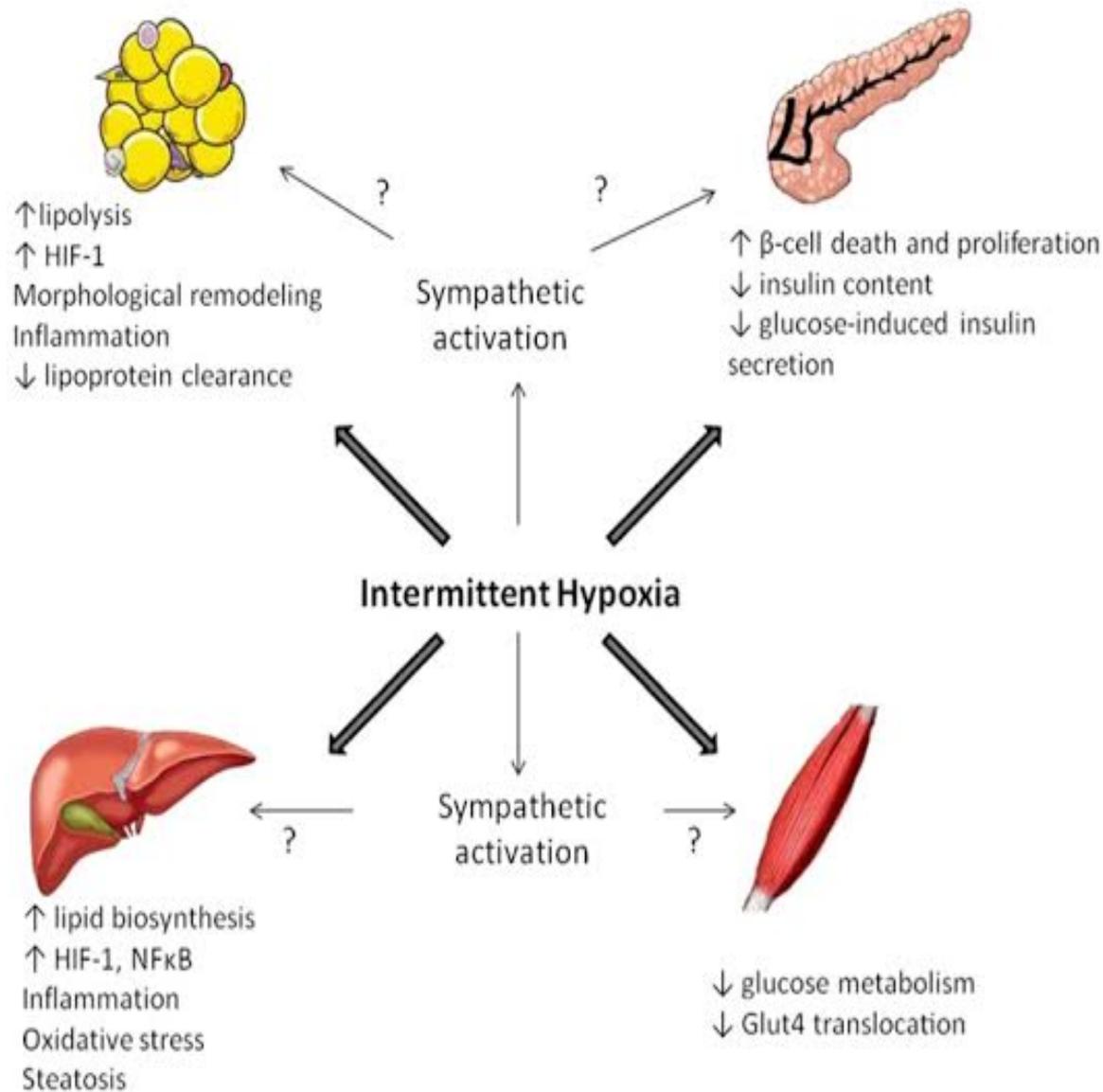


Figure 2 Mechanisms linking intermittent hypoxia to impaired glucose metabolism. Intermittent hypoxia acts on pancreatic insulin production and secretion as well as on insulin target organs such as adipose tissue, liver and skeletal muscle. These combined effects induce impaired glucose tolerance, insulin resistance and dyslipidemia. Intermittent hypoxia effects may be direct and/or mediated through the activation of the sympathetic nervous system. HIF-1 α (hypoxia inducible factor 1-alpha), NF- κ B (nuclear factor- κ B), GLUT4 (glucose transporter type 4).

CORPUS CALLOSUM

The bridge joining the two halves of the brain, called the corpus callosum, is larger in Marge than in Homer. Marge can integrate information from the two halves of the brain, meaning that she can simultaneously make Homer's lunch, listen to Lisa's saxophone playing and insure that Bart doesn't burn anything, while Homer has trouble combining singing and driving without crashing into a chestnut tree.

HEART AND CIRCULATORY SYSTEM

While Marge is young, Bart's shenanigans are less likely to give her high-blood pressure than Homer's. Her higher levels of estrogen prevent cholesterol deposits from forming on artery walls. By the time Lisa graduates from Yale, however, Marge's risk for heart disease will begin to match his.

LYMPHATIC SYSTEM

If Marge gets a cold, her immune system will respond more forcefully than Homer's immune system. But she is also more likely to suffer from diseases, like rheumatoid arthritis and lupus erythematosus, linked to a highly active immune system that malfunctions.

LIVER

Homer metabolizes beer faster and more efficiently than Marge does, so he is less likely to get a hangover. But that immunity may partly explain why more men are alcoholics.

STOMACH

No one can dispute that Homer is the gourmand of the Simpsons. He is unable to resist pork chops, chocolate and Vaseline. But in the unlikely event that he decided to lose weight, he could diet. For Marge, however, losing weight would require not only giving up Jello desserts, but exercising.

SKELETAL SYSTEM

Homer's body will always produce testosterone, but estrogen production virtually halts when a woman goes through menopause. Because these hormones rejuvenate bones, this means that while Homer will always survive cliff falls on Bart's skateboard unscathed, Marge's bones could become more brittle.

SLEEP DISORDERED BREATHING

A Randomized Controlled Trial of Continuous Positive Airway Pressure on Glucose Tolerance in Obese Patients with Obstructive Sleep Apnea

Neus Salord, MD^{1,2,3,4}; Ana Maria Fortuna, MD^{3,4,5}; Carmen Monasterio, MD^{1,2,4}; Mercè Gasa, MD^{1,2}; Antonio Pérez, MD^{3,6,7}; Maria R. Bonsignore, MD⁸; Núria Vilarrasa, MD^{7,9}; Josep Maria Montserrat, MD^{4,10}; Mercedes Mayos, MD^{3,4,5}

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Study Objectives: Obstructive sleep apnea (OSA) is associated with an increased prevalence of metabolic syndrome (MetS), even in patients with morbid obesity. Our goal was to address whether continuous positive airway pressure (CPAP) treatment improved glucose metabolism in this population.

Methods: A prospective randomized controlled trial was performed in severe OSA patients with morbid obesity without diabetes in two university referral hospitals. Patients received conservative (CT) versus CPAP treatment for 12 weeks. MetS components, homeostasis model assessment of insulin resistance (HOMA-IR) and oral glucose tolerance were assessed at baseline and after treatment.

Results: A total of 80 patients completed the study (42 CPAP and 38 CT patients). After 12 w of CPAP treatment, weight loss was similar in both groups and physical activity, prevalence of MetS, and HOMA-IR did not change in either group. In the CPAP group impaired glucose tolerance (IGT) reversed in nine patients and IGT developed in none, whereas IGT reversed in five patients and IGT developed in five patients in the CT group ($P = 0.039$ in the Fisher test). Changes in 2-h plasma glucose after glucose load were greater in the CPAP group than in the CT group (CPAP: -0.5 ± 1.5 versus CT: 0.33 ± 1.9 , $P = 0.007$).

Conclusions: The improvement of glucose tolerance in morbidly obese patients with severe obstructive sleep apnea, without changes in homeostasis model assessment of insulin resistance, supports an improvement in peripheral insulin resistance after continuous positive airway pressure treatment.

Clinical Trials Registration: NCT 01029561.

Keywords: continuous positive airway pressure, glucose tolerance, insulin resistance, obstructive sleep apnea

Citation: Salord N, Fortuna AM, Monasterio C, Gasa M, Pérez A, Bonsignore MR, Vilarrasa N, Montserrat JM, Mayos M. A randomized controlled trial of continuous positive airway pressure on glucose tolerance in obese patients with obstructive sleep apnea. *SLEEP* 2016;39(1):35–41.

- Es el primer estudio que investiga el efecto de la CPAPn sobre el meta. de la glucosa en población específica .
- Los SAHS grave con obesidad mórbida en tratamiento con CPAPn durante doce semanas ,mejoraban la tolerancia a la glucosa .
- Estos resultados apoyan la mejoría de la resistencia periférica a la insulina después del tratamiento con CPAPn

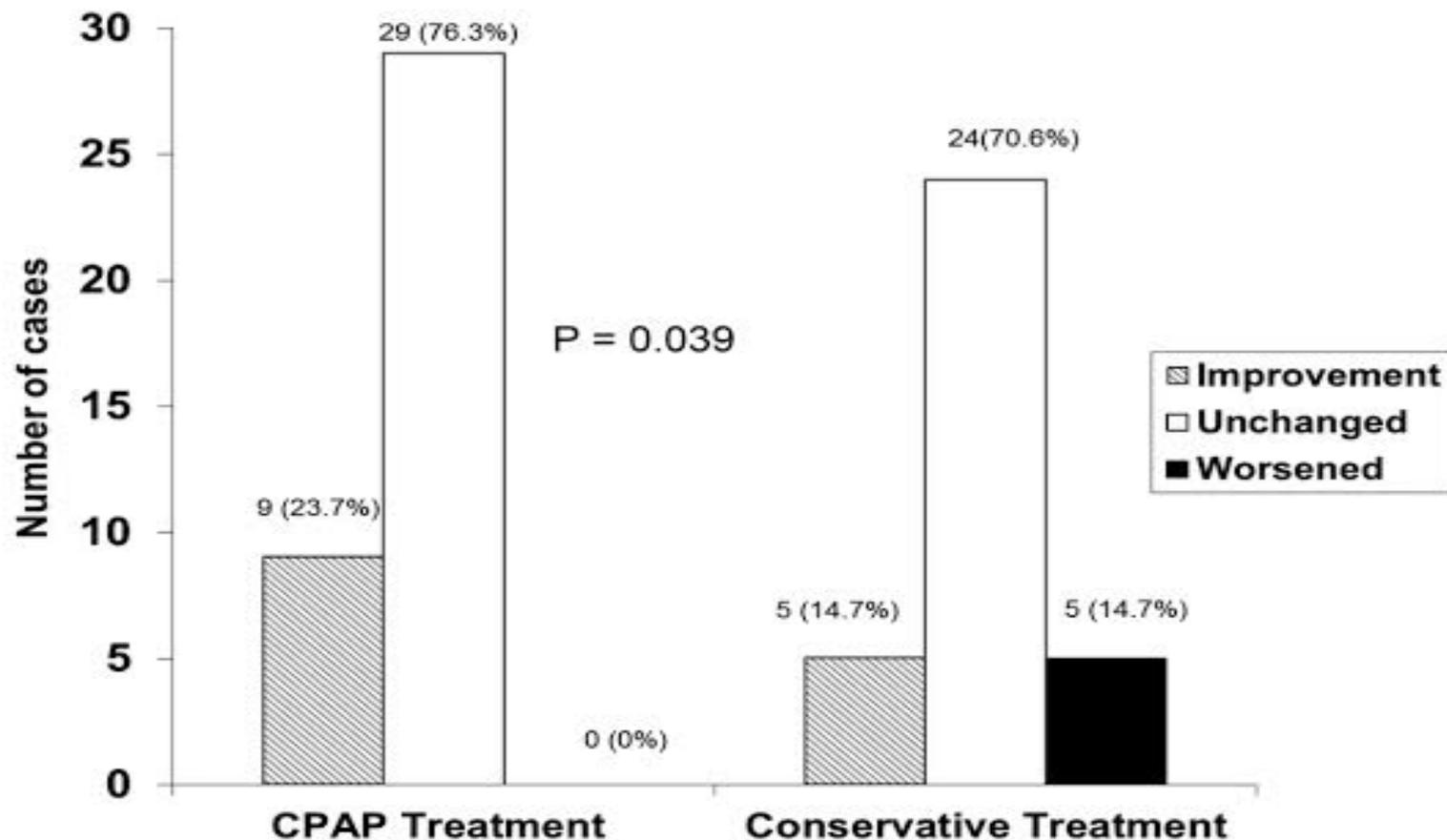


Figure 2—Effect of 12 w continuous positive airway pressure and conservative treatment on glucose tolerance in subjects with morbid obesity and severe sleep apnea. Improvement was considered if a patient with glucose intolerance had normal tolerance at the end of the study. Worsened was considered if a patient with normal tolerance turned to intolerant at the end of the study.

ORIGINAL ARTICLE

Eight Hours of Nightly Continuous Positive Airway Pressure Treatment of Obstructive Sleep Apnea Improves Glucose Metabolism in Patients with Prediabetes

A Randomized Controlled Trial

Sushmita Pamidi¹, Kristen Wroblewski², Magdalena Stepień³, Khalid Sharif-Sidi³, Jennifer Kilkus³, Harry Whitmore³, and Esra Tasali³

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- Demuestran que 8 horas de tratamiento con CPAPn durante dos semana reduce la respuesta general al test de tolerancia a la glucosa oral y mejora la sensibilidad a la insulina .
- Utilizar 8 horas diarias de CPAPn proporciona a los paciente prediabeticos beneficios cardiometabolicos y puede prevenir complicaciones cardiovasculares.
- Parece que hay una correlación positiva entre las horas de uso de CPAPn y los cambios metabólicos

ORIGINAL ARTICLE

Effects of CPAP on body weight in patients with obstructive sleep apnoea: *a meta-analysis of randomised trials*

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Isabela M Benseñor,^{1,3} Paulo A Lotufo^{1,3}

- Incluyeron 3.181 pacientes de 25 ensayos randomizados
- Este estudio resalta que la introducción del CPAPn promueve un pequeño pero significativo aumento del IMC y peso
- El incremento del peso en SAHS después del tto con CPAPn no está influenciado por la edad, género, IMC, peso, severidad del OSA, el diseño del estudio, duración y cumplimiento de la CPAP o la presencia de dieta o actividad física

HIPOTESIS

- Posible cambio en las hormonas que controla la saciedad y el hambre (orexinas).
- El CPAPn restaura sueño profundo y por lo tanto incrementa el anabolismo.
- SAHS activa la lipólisis de tejido adiposo y favorecería la pérdida de peso .
- El SAHS produce un mayor gasto energético



- El tratamiento con CPAPn a largo plazo produce una redistribución de grasa intraabdominal.
- En los pacientes tratados con La CPAPn disminuye el tejido adiposo subcutáneo sin cambios en la grasa intraabdominal preaórtica (PIF). En los pacientes no tratados con CPAPn aumenta el PIF.
- Estos hallazgos apoyan que el tratamiento con CPAPn se asocia con un perfil antropométrico de menor riesgo cardiovascular.
- Utilización de la ecografía

NEUMOLOGO
DE GUARDIA

GRASA SUBCUTANEA,
GRASA PERIVISCERAL....
ROSQUILLAAAASSS....
DESAYUNO..AAAHHH!!!!



ORIGINAL ARTICLE

Associations between Obstructive Sleep Apnea, Sleep Duration, and Abnormal Fasting Glucose

The Multi-Ethnic Study of Atherosclerosis

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- Encuentra una asociación independiente en los SAHS moderados/severos entre obesidad , duración del sueño y niveles de glucosa en ayunas en población Afroamericana y raza blanca.
- Por el contrario esta relación es mucho mas débil en subgrupos Hispanos y Asiáticos.
- Sugiriendo que la etnicidad puede modificar el efecto del SAHS en los niveles de glucosa en ayunas .



CrossMark

Intermittent hypoxia alters gut microbiota diversity in a mouse model of sleep apnoea

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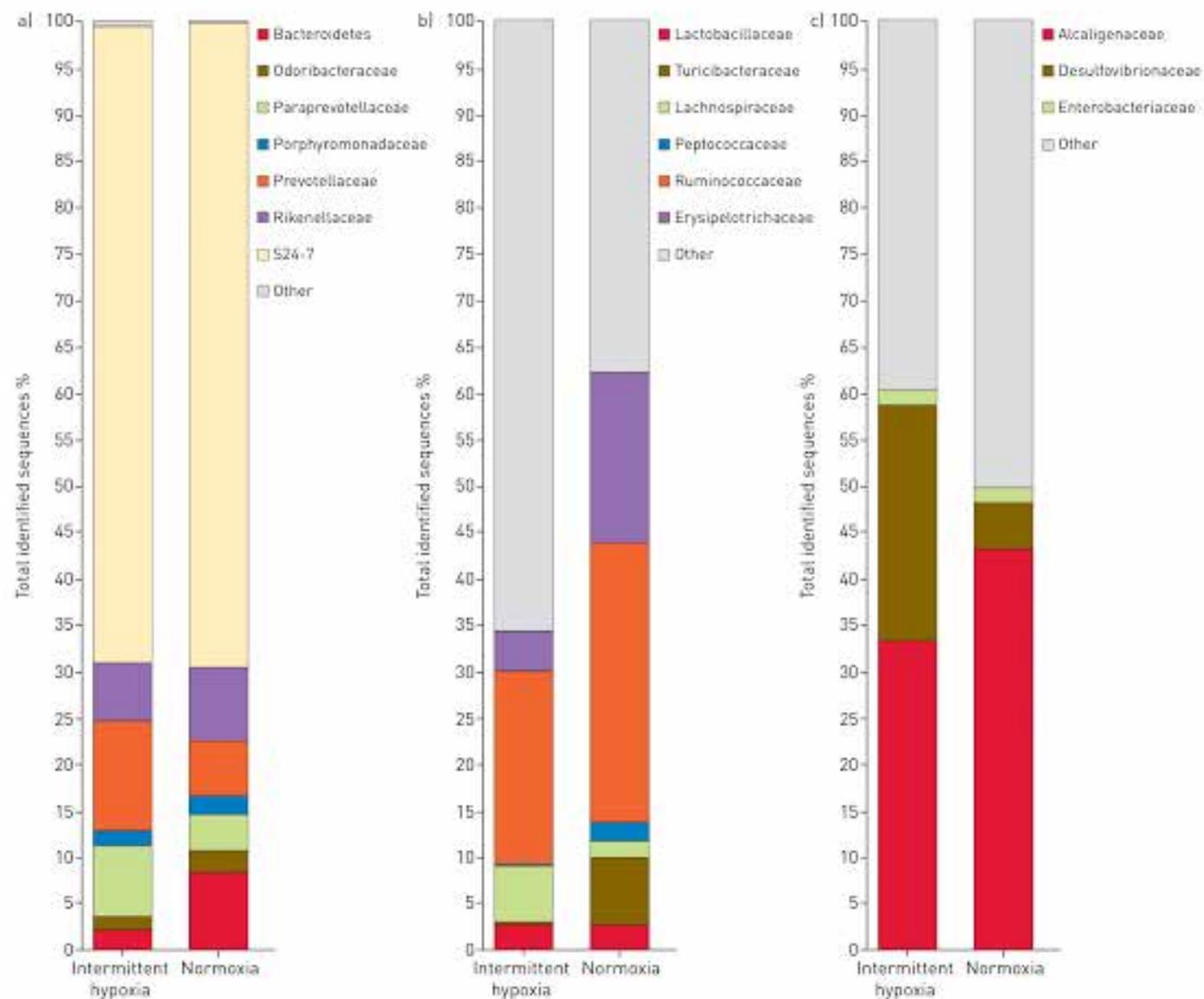


FIGURE 5 Family level microbial classification of bacteria from intermittent hypoxia and normoxia faecal samples. a) Bacteroidetes family, b) Firmicutes family and c) Proteobacteria family.

- Los ciclos de hipoxia y reoxigenación del SAHS podría modificar el microbiana intestinal .
- Las alteraciones en la flora intestinal inducida por HI podría ser relevantes en las alteraciones metabólicas, obesidad y síndrome metabólico

- Se sugiere que la relación de equilibrio del microbioma intestinal puede verse comprometido en pacientes con SAHS.
- Se abre un campo en la investigación de cómo la HI y/o fragmentación del sueño, puede afectar al microbioma intestinal e influir sobre el metabolismo del sujeto.

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DIRECTOR: FRED ZINNEMANN**

Impact of obstructive sleep apnoea on insulin resistance in nonobese and obese children

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The puzzle of metabolic effects of obstructive sleep apnoea in children



CrossMark

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- La obesidad , como la fragmentación del sueño se asocian a alteraciones metabólicas incluyendo la resistencia a la insulina y el perfil lipídico.
- La gravedad de la hipoxemia nocturna ejerce un efecto menor.
- Es el mayor estudio publicado sobre los efectos metabólicos en niños y tienen que considerarse como uno de los mas importantes



GRACIAS