Effect of CPAP on the metabolic syndrome: a randomised sham-controlled study

A recently published editorial[1] concluded that severity of disease, Continuous Positive Airway Pressure (CPAP) compliance and comorbidities might explain discrepancies between a randomised sham-controlled crossover study[2] which showed that CPAP reversed metabolic syndrome (metS) and reduced weight, body mass index (BMI) and visceral abdominal fat and our findings from a randomised sham-controlled parallel-group study.[3] Whether CPAP might be a novel method to reverse metS in those with Obstructive Sleep Apnea (OSA) is an intriguing possibility, since diagnosing and treating metS is important.[1] We omitted to examine the effect of CPAP on metS in our

Table 1 The development and regression of metS from baseline to week 12

	CPAP		Sham	
	metS	n MetS	metS	n MetS
Baseline	18	14	14	17
Week 12				
metS	12	2	8	3
n MetS	3	10	1	10

Data are n metS, metabolic syndrome.

Table 2 Randomised sham-controlled studies examining the effect of CPAP on visceral abdominal fat (VAF)

	n							
	M	F	Design	AHI	BMI	Duration (weeks)	CPAP effect on VAF	CPAP effect on BMI
Hoyos *et al*[3]	65	0	Parallel	40	31	12	−0.06 (−0.58 to 0.70) p=0.85	0.07 (−0.11 to 0.26) p=0.79
Sharma *et al*[2]	77	9	Cross-over	50	33	12	−0.20 (−0.37 to −0.06)* p=0.01	−0.06 (−0.1 to −0.01)* p<0.001
Sivam *et al*[6]	26	1	Cross-over	57	31	8	−0.03 (−0.15 to 0.08) p=0.59	0.07 (−0.05 to 0.05) p=0.32
Kritikou *et al*[7]	22	20	Cross-over	42	27	8	0.14 (−0.09 to 0.37) p=0.25	0.07 (−0.24 to 0.38) p=0.67
				32	30		0.08 (−0.28 to 0.45) p=0.66	0.18 (−0.07 to 0.43) p=0.17

Data are calculated standardised effect sizes (95% CI) after treatment, unless otherwise stated.
*Values are adjusted for baseline.
M, Male; F, Female; AHI, Apnea Hypopnea Index; BMI, body mass index.

population, a typical OSA cohort with treated long-standing metabolic comorbidites and less than ideal CPAP usage.[1] To rectify this, we retrospectively assayed stored blood for lipids and abstracted information regarding hypertension, hyperlipidaemia and its treatment to diagnose metS.

The study design and baseline characteristics have been previously reported.[3] MetS was defined according to international consensus guidelines,[4] and the presence (or absence) of metS was assessed at 0 and 12 weeks. The change in the proportion of participants with or without metS from baseline were analysed by generalised linear models examining the treatment by time interaction (SAS V.9.2). Analyses utilised generalised estimating equations and an exchangeable correlation structure, which were then confirmed by Bayesian methods.

Reversal of metS after 12 weeks occurred in 3 of 18 (17%) men with metS at baseline treated with CPAP compared with 1 of 14 (7%) men treated with sham; whereas metS developed in 2 of 14 (14%) men without metS at baseline compared with 3 of 17 (18%) men treated with sham (time by treatment interaction p=0.28): table 1. This indicates that 12 weeks of CPAP therapy had no effect on the development or regression of metS. Utilising Bayesian methods, restricting the analysis to the 49 men with complete data, or using the original National Cholesterol Education Program Adult Treatment Panel III criteria for diagnosing metS did not alter this finding.

CPAP therapy remains the standard care for OSA, however its effect on metS has only been previously examined in two contradictory randomised cross-over studies,[2 5] and now by us. On the other hand, all randomised sham-controlled studies show no effect of CPAP on visceral abdominal fat, BMI and weight, except one[2]: table 2. Our original report and these additional data support the conclusion[1] that CPAP is unlikely to have a major effect on metabolic health in unselected individuals with OSA.

CM Hoyos,[1] DR Sullivan,[2] PY Liu[1,3]

[1]Endocrine and Cardiometabolic Research Group, NHMRC Centre for Integrated Research and Understanding of Sleep (CIRUS), Woolcock Institute of Medical Research, University of Sydney, Sydney, New South Wales, Australia
[2]Biochemistry Department, Royal Prince Alfred Hospital, Sydney, New South Wales, Australia
[3]Division of Endocrinology, Department of Medicine, David Geffen School of Medicine at UCLA, Harbor-UCLA Medical Center and Los Angeles Biomedical Research Institute, Torrance, California, USA

Correspondence to Dr Peter Y Liu, Division of Endocrinology, Department of Medicine, David Geffen School of Medicine at UCLA, Harbor-UCLA Medical Center and Los Angeles Biomedical Research Institute, 1124 W. Carson Street, Torrance, CA 90502, USA; pliu@labiomed.org

Contributors Study concept and design: PYL; Acquisition of data: CMH, DRS; Analysis and interpretation of data: CMH, PYL; Drafting of the manuscript: CMH, PYL; Critical revision of the manuscript: CMH, DRS, PYL; Statistical analysis: CMH PYL; Obtained funding: PYL.

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