treatment. This may be an important, yet frequently overlooked factor, partly explaining, along with the short follow-up period, why most studies failed to show a significant effect of CPAP treatment on glycaemic control and metabolic profile of OSA patients. Indeed, in the studies included in this article that have examined this parameter, there was a considerable variation of mean CPAP use (range 3.6–6.6 $h \cdot night^{-1}$).

Moreover, only two randomised studies exist in the literature comparing the effects of therapeutic *versus* subtherapeutic CPAP treatment [2, 3]. In both studies, the average nocturnal therapeutic CPAP use was <4 h. Specifically, it was 3.6 h in the study by WEST *et al.* [2] and 3.9 h in the study by COUGHLIN *et al.* [3]. Hence, one may question whether insufficient CPAP use is a potential confounding factor in their negative findings.

Conversely, we have recently demonstrated that CPAP use for >4 h per night is crucial in ameliorating HbA_{1c} and total cholesterol levels along with several inflammation markers after 6 months of CPAP treatment in nondiabetic OSA patients [4, 5]. Of note, it was exclusively shown in adherent patients that CPAP treatment had a beneficial metabolic effect [4, 5].

Clearly, the role of adherence to CPAP therapy in these inconsistent results should be highlighted rather than underestimated. Given that there is no consensus on the minimum duration of CPAP use for a beneficial metabolic effect, further research is eagerly awaited to establish the optimal use of this modality for ameliorating the metabolic consequences in OSA patients.

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REFERENCES

1 Lévy P, Bonsignore MR, Eckel J. Sleep, sleep-disordered breathing and metabolic consequences. *Eur Respir J* 2009; 34: 243–260.
2 West SD, Nicoll DJ, Wallace TM, *et al.* The effect of CPAP on insulin resistance and HbA1c in men with obstructive sleep apnoea and type 2 diabetes. *Thorax* 2007; 62: 969–974.
3 Coughlin SR, Mawdsley L, Mugarza JA, *et al.* Cardiovascular and metabolic effects of CPAP in obese men with OSA. *Eur Respir J* 2007; 29: 720–727.
4 Steiropoulos P, Tsara V, Nena E, *et al.* Effect of continuous positive airway pressure treatment on serum cardiovascular risk factors in patients with obstructive sleep apnea-hypopnea syndrome. *Chest* 2007; 132: 843–851.
5 Steiropoulos P, Papanas N, Nena E, *et al.*, Markers of glycemic control and insulin resistance in non-diabetic patients with obstructive sleep apnea hypopnea Syndrome: does adherence to CPAP treatment improve glycemic control? *Sleep Med* 2009; 10: 887–891.

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From the author:

In response to our recent article on sleep, sleep-disordered breathing and metabolism [1], P. Steiropoulos and co-workers wish to emphasise the role of adherence to continuous positive airway pressure (CPAP) treatment in improving obstructive sleep apnoea (OSA) metabolic status. This is certainly a major factor of success regarding CPAP treatment effectiveness. Whilst CPAP compliance remains a major challenge [2], the optimal duration of treatment remains unclear. It has been suggested that improving vigilance and cognitive function would need a minimum of 5 h, apparently with additional benefits when the duration of treatment was further increased [3]. This is much less clear as regard cardiovascular and metabolic changes. There is apparently a relationship between CPAP duration and reduction in blood pressure [4]. There is very limited evidence regarding CPAP effects on glycaemic control. P. Steiropoulos and co-workers noticed that mean CPAP use in the two randomised control trials was <4 h, 3.6 [5] and 3.9 h [6], respectively. Thus, they question whether insufficient CPAP use may be a potential confounding factor in the published negative findings. They have recently demonstrated that CPAP use for >4 h per night is crucial in ameliorating HbA_{1c} and total cholesterol levels along with several inflammation markers after 6 months of CPAP treatment in non-diabetic OSA patients, with only adherent patients exhibiting beneficial metabolic effect [7]. They also recently reported that only patients using CPAP for >4 h had significant reduction in soluble and cellular immune response factors [8]. Their study on glycaemic control [7] is not a randomised controlled trial and as such has significant limitations. Moreover, good adherence to long-term CPAP treatment seems to significantly reduce HbA_{1C} levels but has no effect on markers of insulin resistance [7]. The study by HARSCH *et al.* [9] did not shown any effect on insulin sensitivity in OSA patients with a body mass index >30 $kg \cdot m^{-2}$. In this study, the mean compliance to CPAP was high, *i.e.* 5.2 ± 0.91 h [9]. In addition, there was no association between mean duration of CPAP use per night and change in insulin sensitivity from baseline to 3 months [9]. Insulin resistance and glycaemia are closely linked in obesity and diabetes pathophysiology. Thus, it may be concluded that CPAP compliance is certainly an important issue. However, obesity is a major confounding factor in OSA. Thus, it is not surprising that CPAP effects on glycaemic control could be modulated by the degree of obesity. It should certainly be further studied by large randomised controlled trials [10, 11].

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REFERENCES

1 Lévy P, Bonsignore MR, Eckel J. Sleep, sleep-disordered breathing and metabolic consequences. *Eur Respir J* 2009; 34: 243–260.

2 Pepin JL, Krieger J, Rodenstein D, *et al.* Effective compliance during the first 3 months of continuous positive airway pressure. A European prospective study of 121 patients. *Am J Respir Crit Care Med* 1999; 160: 1124–1129.
3 Weaver TE, Maislin G, Dinges DF, *et al.* Relationship between hours of CPAP use and achieving normal levels of sleepiness and daily functioning. *Sleep* 2007; 30: 711–719.
4 Haentjens P, Van Meerhaeghe A, Moscariello A, *et al.* The impact of continuous positive airway pressure on blood pressure in patients with obstructive sleep apnea syndrome: evidence from a meta-analysis of placebo-controlled randomized trials. *Arch Intern Med* 2007; 167: 757–764.
5 West SD, Nicoll DJ, Wallace TM, *et al.* Effect of CPAP on insulin resistance and HbA1c in men with obstructive sleep apnoea and type 2 diabetes. *Thorax* 2007; 62: 969–974.
6 Coughlin SR, Mawdsley L, Mugarza JA, *et al.* Cardiovascular and metabolic effects of CPAP in obese males with OSA. *Eur Respir J* 2007; 29: 720–727.
7 Steiropoulos P, Papanas N, Nena E, *et al.*, Markers of glycemic control and insulin resistance in non-diabetic patients with obstructive sleep apnea hypopnea syndrome: does adherence to CPAP treatment improve glycemic control? *Sleep Med* 2009; 8: 887–891.
8 Steiropoulos P, Kotsianidis I, Nena E, *et al.* Long-term effect of continuous positive airway pressure therapy on inflammation markers of patients with obstructive sleep apnea syndrome. *Sleep* 2009; 32: 537–543.
9 Harsch IA, Schahin SP, Radespiel-Troger M, *et al.* Continuous positive airway pressure treatment rapidly improves insulin sensitivity in patients with obstructive sleep apnea syndrome. *Am J Respir Crit Care Med* 2004; 169: 156–162.
10 Shaw JE, Punjabi NM, Wilding JP, *et al.* Sleep-disordered breathing and type 2 diabetes: a report from the International Diabetes Federation Taskforce on Epidemiology and Prevention. *Diabetes Res Clin Pract* 2008; 81: 2–12.
11 Tasali E, Mokhlesi B, Van Cauter E. Obstructive sleep apnea and type 2 diabetes: interacting epidemics. *Chest* 2008; 133: 496–506.

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