Obstructive sleep apnoea syndrome and cardiovascular risk

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Zamarrón *et al*. [1] report that obstructive sleep apnoea syndrome (OSAS) is associated with raised levels of markers of endothelial function. Moreover, some of these markers (intercellular cell adhesion molecule-1 (ICAM-1) and plasminogen activator inhibitor-1 (PAI-1)) improved after nasal continuous positive airway pressure [1]. Some additional comments may be of interest.

Metabolic syndrome (MetS) is highly prevalent in patients with OSAS [2]. In turn, intermittent hypoxia increases insulin resistance [2]. Consequently, it is difficult to identify the contribution of OSAS and MetS to vascular risk, since they share common predisposing factors (e.g. obesity [2] and endothelial dysfunction [3]). Markers of endothelial activation, including ICAM-1, PAI-1 and E-selectin, are raised in MetS [3] and Zamarrón *et al.* [1] report a similar pattern in OSAS. Platelet activation occurs in MetS due to reduced availability of endothelium-derived nitric oxide (NO) and prostacyclin [4]. Similarly, in OSAS a decrease in NO availability and prostacyclin/thromboxane metabolite ratio has been reported [2, 5]. Markers of platelet activation, including mean platelet volume (MPV) and platelet distribution width (PDW), were increased in OSAS, in parallel with the severity of hypoxia [6]. Therefore, it would be helpful to know how many patients in the control and OSAS group had MetS in the Zamarrón *et al.* [1] study.

Taken together, all these findings highlight the role of OSAS on endothelial integrity and as a contributor to cardiovascular risk.

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