

Letters to the Editor

Circulating Microparticles From Patients With Coronary Artery Disease Cause Endothelial Dysfunction

Micropartículas circulantes de pacientes con enfermedad coronaria causan disfunción endotelial

To the Editor,

In a recent article in *Revista Española de Cardiología*,¹ the author considered the debate about the impact of microparticles (MP), specially ultrafine ones, on the onset of acute coronary syndrome (ACS). However, in order to determine what the exact role of these particles is in the genesis of ACS, as well as to quantify the added risk and its effect on risk stratification of patients with ACS, the authors did not discuss the markers currently available. These are based on demonstrating the presence of endothelial dysfunction associated with this condition. Both experimental and clinical studies have indicated that endothelial dysfunction can be considered as a biomarker, as such dysfunction has been analyzed in different studies as an independent predictor of adverse events occurring in patients diagnosed with ACS.^{2,3} To this end, plasma levels of CD31+/annexin V+ microparticles have been quantified as a marker of endothelial dysfunction.^{4,5} This same marker has been assessed recently.⁶ An increase in plasma levels has been considered a risk factor for cardiovascular disease with worse endothelial function, itself considered an independent risk factor for adverse cardiovascular events in patients diagnosed with stable ACS. Similarly, Nozaki et al.⁷ studied concentrations of CD144+ as a marker of endothelial dysfunction in a strategy using different biomarkers, such as B natriuretic peptide, highly sensitive C reactive protein, and the quantity of circulating microparticles. These give an indication of endothelial dysfunction in a population at high risk of experiencing ACS. The authors found that high MP levels are an independent predictor of cardiovascular death and ACS. This biomarker also provides better stratification of cardiovascular risk and allows patients to benefit from more aggressive therapy that might improve their prognosis. Endothelial dysfunction caused by circulating MP is a risk factor for

cardiovascular disease and can be added to the risk factors considered as “traditional.” Different biomarkers can be measured, and confirm the importance of these MP in the genesis of ACS.

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Circulating Microparticles From Patients With Coronary Artery Disease Cause Endothelial Dysfunction. Response

Micropartículas circulantes de pacientes con enfermedad coronaria causan disfunción endotelial. Respuesta

To the Editor,

I would like to thank the interesting comments of Bueno Jiménez et al. published in *Revista Española de Cardiología* in relation to the Editorial article¹ where I discussed the plausibility of the exposure to ambient ultrafine particles (UFP) as a risk factor for cardiovascular diseases. Numerous epidemiological studies support the association of exposure to ambient particulate matter (PM) with cardiovascular ischemic endpoints² and substantial experimental animal work support the feasibility for causality in

the case of atherosclerosis.³ While it is likely that PM with a smaller particle size such as UFP carry greater cardiovascular toxicity than particles with larger size, the possibility that inhaled UFP could translocate and access the systemic circulation still awaits more definite confirmation.³ Ambient PM can lead to systemic vascular effects by several hypothetical mechanisms that still remain to be elucidated.¹ The author's comment about the need to employ novel biomarkers for endothelial dysfunction, such as circulating endothelium-derived microparticles (EMP), that can better capture the cardiovascular risk potentially attributable to ambient PM and UFP is very appropriate as they could also aid in the identification of pathogenic mechanisms. EMPs, characterized by different phenotypes such as CD31⁺/CD41[−], CD31⁺/annexin V⁺, CD144⁺ and/or CD62e⁺ appear not only to reflect endothelial dysfunction and serve as an stratifying biomarker⁴ but also to exert pathogenic actions such as procoagulant