Microparticles: An Introduction

Chantal M. Boulanger, Françoise Dignat-George

The notion that cells shed small membrane vesicles from their plasma membrane was reported some 40 years ago, and for many years this process was described as the release of “cell dust.” However, the past decade has seen an unprecedented interest in membrane vesicles in many fields of biology, including vascular biology and thrombosis. Consequently, the term cell dust has been replaced by the term microparticles to describe submicron membrane vesicles shed from activated or apoptotic cells. Exciting findings have further demonstrated the unexpected biological impact of these microparticles on cellular crosstalk and their contribution to inflammation, thrombosis, and angiogenesis. A thorough investigation of the mechanisms governing their release and their interaction with target cells is now required for a proper understanding of their contribution to disease progression or repair mechanisms.

The review series begins with an article by Morel et al summarizing the state of the art on the mechanisms governing the formation of circulating microparticles. Platelet-derived microparticles represent one of the major populations of circulating microparticles and studies on their generation have greatly contributed to our knowledge of the mechanisms involved in the loss of membrane phospholipid asymmetry. Interestingly, Scott’s syndrome is a rare congenital bleeding disorder that presents as a defect in platelet membrane remodeling. Morel et al also draw our attention to the role of intracellular calcium concentration in cytoskeleton reorganization during aminophospholipid externalization. Whether or not these mechanisms regulating membrane and cytoskeleton remodeling in platelets also hold true for microparticle formation in other cell types remains to be established. The pathophysiological relevance of microparticle formation is highlighted in the review by Zwicker et al, that focuses on the contribution of microparticles bearing tissue factor to thrombus formation. The notion of a “blood-borne tissue factor” conveyed by circulating microparticles represents an alternative mechanism for activation of blood coagulation. However, much debate still exists regarding the presence of tissue factor activity on circulating microparticles, despite the general consensus that tissue factor antigen is measurable in plasma. The third review article of this series focuses on microparticles of endothelial origin. Although they represent a minor population of the overall pool of circulating microparticles, endothelial microparticles present a surrogate marker of activated/apoptotic endothelial cells. Depending on the cause of endothelial activation, they display specific phenotypes that could contribute to their multifaceted effects on vascular homeostasis.

The next set of reviews addresses and reviews the role of microparticles in the development of cardiovascular diseases and potential repair mechanisms. Tushuizen et al discuss the potential deleterious or beneficial effects of circulating microparticles on the vessel wall. They also highlight the notion that plasma levels of specific subpopulations of microparticles are valuable surrogate
markers of vascular health and that evidence suggests that they have potential prognostic value in cardiovascular diseases. Finally, Shai and Varon discuss the contribution of microparticles to tissue repair and pathological angiogenesis, such as cancer angiogenesis or diabetic retinopathy. The mechanisms governing these microparticle effects involve direct activation of signaling pathways, transfer of growth factors or new transcripts, or pericellular proteolytic activities mediated by serine proteases and metalloproteases.

This review series on microparticles will introduce *Arteriosclerosis, Thrombosis, and Vascular Biology* readers to the field of shed-membrane vesicles and their wide spectrum of actions in cardiovascular homeostasis. The ubiquitous formation of these vesicles certainly raises questions regarding their unforeseen contribution to a large number of biological responses. A better understanding of mechanisms governing their generation and of their potential to reprogram remote cells in pathological conditions represents a new dimension in cellular communication.
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