Microparticles harboring Sonic Hedgehog promote angiogenesis through the upregulation of adhesion proteins and proangiogenic factors

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Résumé en anglais	Microparticles (MPs) are small fragments generated from the plasma membrane after cell stimulation or apoptosis. We have recently shown that MPs harboring the morphogen Sonic Hedgehog (MPsShh+) correct endothelial injury by release of nitric oxide from endothelial cells [Agouni, Mostefai, Porro, Carusio, Favre, Richard, Henrion, Martínez and Andriantsitohaina (2007) FASEB J., 21, 2735–2741]. Here, we show that MPsShh+ induce the formation of capillary-like structures in an in vitro model using human endothelial cells, although they inhibited cell migration. Besides, MPsShh+ regulate cell proliferation. Both cell adhesion and expression of proteins involved in this process such as Rho A and phosphorylation of focal-activated kinase were increased by MPsShh+, via a Rho-associated coiled-coil-containing protein kinase inhibitor-sensitive pathway. We demonstrate that MPsShh+ increase messenger RNA and protein levels of proangiogenic factors as measured by quantitative reverse transcription–polymerase chain reaction and western blot. In spite of vascular endothelial growth factor expression, conditioned media from endothelial cells treated avec MPsShh+ reduces angiogenesis. Interestingly, the effects induced by MPsShh+ on the formation of capillary-like structures, expression of adhesion molecules and proangiogenic factors were reversed after silencing of the Shh receptor, using small interfering RNA or when Sonic Hedgehog (Shh) signaling was pharmacologically inhibited with cyclopamine. Taken together, we show that Shh carried by MPsShh+ regulate angiogenesis probably through both a direct and an indirect mechanisms, and we propose that MPs harboring Shh may contribute to the generation of a vascular network in pathologies associated with tumor growth.
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