Microparticles from apoptotic monocytes enhance nitrosative stress in human endothelial cells.

Titre	Microparticles from apoptotic monocytes enhance nitrosative stress in human endothelial cells.
Type de publication	Article de revue
Auteur	Mastronardi, Maria Letizia [1], Mostefai, Hadj Ahmed [2], Soleti, Raffaella [3], Agouni, Abdelali [4], Martinez, Maria Carmen [5], Andriantsitohaina, Ramaroson [6]
Editeur	Wiley
Type	Article scientifique dans une revue à comité de lecture
Année	2011
Langue	Anglais
Date	2011 Dec
Numéro	6
Pagination	653-660
Volume	25
Titre de la revue	Fundamental and Clinical Pharmacology
ISSN	1472-8206
Mots-clés	Apoptosis [7], Caveolin 1 [8], Cell Line [9], Cell-Derived Microparticles [10], Endothelial Cells [11], Gene Expression Regulation [12], Humans [13], Mitogen-Activated Protein Kinase 1 [14], Mitogen-Activated Protein Kinase 3 [15], Monocytes [16], Nitric oxide [17], Oxidative Stress [18], Phosphatidylinositol 3-Kinases [19], Signal Transduction [20]

Résumé en anglais	Microparticles are membrane vesicles with procoagulant and proinflammatory properties released during cell activation or apoptosis. Microparticles from monocytes have been implicated in atherosclerosis and vascular inflammation, but their direct effects on endothelial cells are not completely elucidated. The present study was designed to dissect the signaling pathways of monocytic microparticles in endothelial cells with respect to both NO pathway and reactive oxygen species. Microparticles were produced by treatment of human monocytic cell line THP-1 with the apoptotic agent VP-16. Human endothelial cells were treated with monocytic microparticles and then, we studied their effects on nitrosative and oxidative stresses. Incubation of human endothelial cells with microparticles enhanced the production of NO without affecting superoxide anions generation. Microparticles did not affect endothelial NO synthase expression and its phosphorylation. Interestingly, microparticles decreased caveolin-1 expression and increased its phosphorylation. Inhibition of PI-3-kinase or MEK1/2 reversed the effects of microparticles on caveolin-1 expression but not its phosphorylation. Moreover, microparticles increased nitration of several proteins, reflecting peroxynitrite production, which was prevented by blockade of PI-3-kinase pathway. In summary, monocyte microparticles active multiple pathways related to nitrosative stress in endothelial cells including both PI-3-kinase and ERK1/2 in the regulation of caveolin-1 expression. These data underscore the pleiotropic effect of microparticles on endothelial cells and suggest that they probably play a critical role on vascular function.
URL de la notice	http://okina.univ-angers.fr/publications/ua10803 [21]
DOI	10.1111/j.1472-8206.2010.00898.x [22]
Lien vers le document	http://dx.doi.org/10.1111/j.1472-8206.2010.00898.x [22]
Titre abrégé	Fundam Clin Pharmacol
Identifiant (ID) PubMed	21105910 [23]

Liens

[1] http://okina.univ-angers.fr/publications?f[author]=19066
[2] http://okina.univ-angers.fr/publications?f[author]=19067
[3] http://okina.univ-angers.fr/r.soleti/publications
[4] http://okina.univ-angers.fr/publications?f[author]=471
[5] http://okina.univ-angers.fr/c.martinez/publications
[6] http://okina.univ-angers.fr/r.andrian/publications
[7] http://okina.univ-angers.fr/publications?f[keyword]=1270
[8] http://okina.univ-angers.fr/publications?f[keyword]=6013
[9] http://okina.univ-angers.fr/publications?f[keyword]=7478
[10] http://okina.univ-angers.fr/publications?f[keyword]=6046
[11] http://okina.univ-angers.fr/publications?f[keyword]=6096
[12] http://okina.univ-angers.fr/publications?f[keyword]=9152
[13] http://okina.univ-angers.fr/publications?f[keyword]=991
[14] http://okina.univ-angers.fr/publications?f[keyword]=16995
[15] http://okina.univ-angers.fr/publications?f[keyword]=16996
[16] http://okina.univ-angers.fr/publications?f[keyword]=10245
[17] http://okina.univ-angers.fr/publications?f[keyword]=6048
[18] http://okina.univ-angers.fr/publications?f[keyword]=6203
[19] http://okina.univ-angers.fr/publications?f[keyword]=16997

[20] http://okina.univ-angers.fr/publications?f[keyword]=6050
[21] http://okina.univ-angers.fr/publications/ua10803
[22] http://dx.doi.org/10.1111/j.1472-8206.2010.00898.x
[23] http://www.ncbi.nlm.nih.gov/pubmed/21105910?dopt=Abstract