# Rosiglitazone, a Peroxisome Proliferator-Activated Receptor-γ Agonist, Prevents Microparticle-Induced Vascular Hyporeactivity through the Regulation of Proinflammatory Proteins




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| Résumé en anglais | Microparticles are plasma membrane vesicles with procoagulant and proinflammatory properties. We recently demonstrated that microparticles induce vascular hyporeactivity and evoke up-regulation of proinflammatory protein expression. This study dissected the effect of either in vitro treatment or short-term oral administration of the peroxisome proliferator-activated receptor-γ (PPARγ) agonist, rosiglitazone, on microparticle-induced vascular hyporeactivity of mouse vessels. Microparticles were produced from T cells by actinomycin D treatment. The effects of rosiglitazone on mouse aortic rings incubated with microparticles were investigated. Aortae treated in vitro with rosiglitazone or aortae taken from mice treated by oral administration of the same agonist completely prevented microparticle-induced vascular hyporeactivity in response to U46619 [9,11-dideoxy-11α, 9α-epoxymethanoprostaglandin F2α). These effects of rosiglitazone occurred independently of the presence of endothelium without modifications in blood parameters. The mechanisms involved abrogation of nitric oxide (NO) and prostacyclin overproduction linked to up-regulation of inducible NO-synthase and cyclooxygenase 2 elicited by microparticles. In addition, rosiglitazone treatment reduced the ability of microparticles to evoke increases in interleukin (IL)-6, IL-8, and nuclear factor (NF)-κB transcription, and NF-κB expression and activation. These results suggest that rosiglitazone, via PPARγ activation, counteracts vascular dysfunction associated with increased release of proinflammatory proteins elicited by microparticles. They underscore therapeutic perspective for rosiglitazone in vascular diseases involving enhanced participation of microparticles. |
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