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Solvent Factors are Central to the Enhancement of Endothelial Progenitor Cells Function by Platelets

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Introduction: Recent evidence has suggested the importance of endothelial progenitor cells (EPCs) in the process of repair post vascular injury and that platelets mediate their recruitment to the site of injury, maturation and differentiation. Yet, the mediators of this reaction are unclear. Platelet activation releases microparticles (PMPs)/ and solvent factors which may mediate this reaction. Thus, the aim of our study is to investigate the main mediators improving EPC functional properties and to identify the specific factors involved in this reaction.
Methods: Human EPCs were isolated from donated buffy coats and cultured for 7 days on a traditional fibronectin matrix in one of the following conditions: 1. Alone (control) 2.Co-incubated with platelets 3. Co incubated with platelets activation products: PMPs/ supernatants 4. Co incubated with platelets and FGF/PDGF inhibitor. EPC functional properties were evaluated by their capacity to form colonies and by the expression of mature endothelial markers such as Tie-2 and DiI-Ac-LDL, using FACS analysis.
Results: After 7 days of culture, the capacity to form colonies and the expression of endothelial markers were higher in EPC co incubated with platelets, PMPs or supernatants compared to EPCs alone. Notably differentiation was higher when EPC were incubated with supernatants compared to PMPs or platelets alone. Furthermore, PDGF/FGF inhibitions significantly reduce Endothelial cell markers expression almost to baseline.
Conclusions: This preliminary study implies that platelets enhance EPC functional properties mainly through solvent factors and less by PMPS. Indeed, it seems that direct interaction is not essential for the platelets' effect on EPC functional properties. Two important mediators which enhance EPC's capacity for differentiation are PDGF and FGF. Further study is required to check additional aspects of EPC functional properties in response to platelet products and PDGF/FGF inhibition.