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Extended Abstract
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# Cardiology-2018- MIF Promoted Cardiovascular Angiogenesis via Erk/Mapk Pathway - Ge Cao -West China Hospital of Si Chuan University, China

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## Introduction

Endothelial cells is the most part of cardiovascular angiogenesis, however, there is no curative therapy for endothelial cells repairing in cardiovascular diseases [1]. At present, multiple clinical trials have indicated that endothelial cells dysfunction aggravate development of restenosis post-PCI, moreover, endothelial injury itself is the cause of many cardiovascular diseases, including myocardial infarction and atherosclerosis . Promoting angiogenesis is the basic aim for endothelial repair. The occurrence and development of endothelial injury is a multi-factor, multi-step and multi-gene interactive process. 6uتcLent blood vessels are essential for blood floZ Moreover, angiogenesis increases viability of ischemic myocardium via multiple mechanisms, while VEGF plays an important role in angiogenesis of cardiovascular diseases . Macrophage migration inhibitory factor (MIF) is a very important cytokines within the organism that can promote the occurrence and development of tumors. Studies have found that the change of MIF level were associated with tumor metastasis and malignant potential [6,7], while there was no report on its cardiovascular angiogenesis eٴوect ERK/MAPK pathways were associated with angiogenesis, and PD98059 was a reported specLfic inhibitor of cell permeability and selectivity, which was involved in ERK1/2 pathways [8]. Hus we determined to explore whether the eٴوect of MIF on endothelial cells was associated with ERK/MAPK pathways, and endothelial cells HUVEC cell line was used in our study

As the pivotal part of cardiovascular angiogenesis, endothelial cells dysfunction is the leading cause of cardiovascular diseases. Macrophage migration inhibitory factor (MIF) is a tumor growth factor with important roles in cervical tumor formation, invasion, progression and metastasis. However, there was no report on effect of MIF on endothelial cells is unclear, and it is still unknown whether MIF is associated with angiogenesis of endothelial cells. Our study was focused on the effect of MIF and PD98059 on endothelial cells HUVEC cell line, so as to investigate the influence of MIF on expression of vascular endothelial growth factor (VEGF). We also explored whether MIF will influence angiogenesis of endothelial cells via ERK/MAPK pathways. Endothelial cells HUVEC cells were conventionally cultured, and Western blot were used to detect the expression of MIF, ERK1 and VEGF proteins after HUVEC cells were intervened by MTT and PD98059. Inter-group difference was statistically assessed. Positive expressions of MIF, ERK1 and VEGF were observed in HUVEC cells. Proliferation activity in MIF group gradually increased after 24 h, 48 h or 72 h treatment (P0.05). Expressions of ERK1 and VEGF are involved in the process of endothelial cells HUVEC cell line. MIF is correlated with increased cell proliferation and promoted cardiovascular angiogenesis via ERK/MAPK pathway for MIF or PD98059 treatment. In summary, we aimed to examine expressions of ERK1 and VEGF so as to explore the eٴوect of MIF on endothelial cells and its relationship with ERK/MAPK pathways.

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