中文題目：Cilostazol經由調控RAGE/ NF-κB訊息傳遞路徑抑制高糖造成之血管平滑肌細胞功能失調

英文題目：Cilostazol inhibits hyperglycemia–induced vascular smooth muscle cell dysfunction by modulating RAGE/ NF-κB signaling pathway

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Background:

An emerging body of evidence suggests that high glucose (HG) causes abnormalities in endothelial and vascular smooth muscle cell function (VSMC) and contribute to atherosclerosis. The receptor for advanced glycation end-products (RAGE) has been subsequently characterized as a multiligand receptor of the immunoglobulin superfamily of cell surface receptors. Cilostazol is known as a clinical medicine in treating diabetic vasculopathy by improving HG-induced vascular dysfunction. In this study, we try to investigate whether cilostazol suppression of HG-induced VSMC dysfunction is through RAGE signaling and its possible regulation mechanism.

Methods:

Aortic tissue of human and STZ diabetes mouse were collected and A7r5 mouse VSMC cell line was used in this study.

Results:

First, our result revealed cilostazol decreased RAGE, VCAM-1, ICAM-1, FAK and MMP-2 expression in HG cultured A7r5 cells and also improved proliferation, adhesion and migration of A7r5 cells. Second, the effects of cilostazol were mainly through inhibiting RAGE/NF-ƙ B pathway and HG induced reactive oxygen species (ROS) production.

Conclusion:

We conclude that the data provide an additional mechanism underlying the anti-atherosclerotic effect of cilostazol by influencing RAGE signal and the downstream molecules. Meanwhile, these

result let us gain a greater understanding of the effectiveness and mechanisms that cilostazol involved in improving the HG induced VSMC dysfunction.