## e0208 NERVE GROWTH FACTOR PROMOTE ANGIOGENESIS OF MESENCHYMAL STEM CELLS

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**Objective** To investigate whether nerve growth factor (NGF) can enhance angiogenesis of mesenchymal stem cells (MSCs), and the possible mechanism.
**Methods** MSCs were seeded into matrigel-coated 24-well plates, and cultured with NGF at different concentrations (0 ng/ml, 25 ng/ml, 50 ng/ml, 100 ng/ml, 200 ng/ml) for 24 h, the tube formation of MSCs was observed and photographed using an inverted microscope. K-252a, the specific inhibitor of NGF receptor TrkA, was used to inhibit the tube formation promoted by NGF. Western Blot was applied to compare the VEGF expression between different groups.
**Results** NGF can promote MSCs tube formation in vitro, which was peaked at the concentration of 50ng/ml with its tubular lengths 2.24-fold increased ($p<0.05$), and was attenuated by K-252a. There was no significant difference of the VEGF expression between NGF treated and control group.
**Conclusion** NGF enhanced the ability of MSCs angiogenesis in vitro, and TrkA signal pathway may be involved.

## e0209 PLATELETDERIVED MICROPARTICLES AFFECTS VASCULAR ENDOTHELIAL GROWTH FACTOR EXPRESSION AND THE MECHANISM IN ENDOTHELIAL CELLS

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**Objective** Detect vascular endothelial growth factor induce by platelet-derived microparticles (PMPs) in human umbilical vein endothelial cells (HUVECs) and its downstream transduction pathway. The aim of our study was to discuss the possible mechanism of PMPs effecting on VEGF releasing and its clinical significance in endothelial cells.
**Methods** Applying the method of flow cytometry (FCM) to isolate platelet-derived microparticles (PMPs) from platelet poor plasma (PPP), and stained with fluorescin isothiocyanate monoclonal antibody against CD61, applying flow cytometry (FCM) method to detect the quantification of PMPs, and the tolal protein concentration of PMPs was determined using Semi-automatic Biochemical Analyser. HUVECs were commonly cultured. Different concentrations of PMPs intervene HUVECs and incubate for different time. We used semi-quantitative reverse transcription PCR techniques to measure VEGF, phosphatidylinositol-3 kinase (PI3K), extracellular signal-regulated kinase (ERK) and VEGF type II receptor (KDR) mRNA levels.
**Results** 1. High density of PMPs can be obtained from PPP after activation of ADP, Thrombin and detected by FCM. 2. Different concentration of PMPs intervene HUVECs, VEGF mRNA level in control group (non intervention group) was significantly higher than the four different concentration of PMPs groups ($p<0.05$, respectively). In contrast, KDR mRNA level in control group (non intervention group) was significantly lower than the four different concentration of PMPs groups ($p<0.05$, respectively). ERK mRNA level in 50 μg/ml PMPs group was significantly higher than the other four groups (1.141 vs 0.749, $p=0.004$), PI3K mRNA level in 100 μg/ml PMPs group was significantly higher than the other groups (1.344 vs 0.999, $p=0.004$). 3. Just as the effect of different concentration PMPs on mRNA expression, VEGF mRNA level in control group (non intervention group) was significantly higher than the 24 h intervention time group (0.318 vs 0.746, $p<0.001$). KDR mRNA level in the 4 h and 24 h groups were significantly higher than control group ($p<0.05$, respectively). PI3K mRNA level in 24 h group was significantly higher than the control group (2.622 vs 0.999, $p=0.004$).
**Conclusions** Abundant PMPs can be obtained from PPP after activation. PMPs may induce the biological processes of blood vessel and angiogenesis via VEGF and its downstream signal transduction pathways.

## e0210 CILOSTAZOL REDUCES NEOINTIMAL HYPERPLASIA BY INHIBITION SUPEROXODE PRODUCTION AND EXPRESSION OF LECTIN-LIKE OXIDISED LDL RECEPTOR-1 AFTER BALLOON COMMON ARTERIAL INJURY IN A RAT MODEL

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Lectin-like oxidised low-density lipoprotein receptor-1 (LOX-1) is a membrane protein that can support the binding, internalisation, and proteolytic degradation of oxidised low-density lipoprotein. The LOX-1 expression and superoxide generation increases in the neointima after balloon injury. Cilostazol, a well-know phosphodiesterase type 3(PDE3) inhibitor for the treatment of peripheral arterial disease, has vasodilator properties and an anti-proliferative action on the growth of vascular smooth muscle cells. To investigate whether cilostazol suppresses intimal hyperplasia and to elucidate its mechanism, we examined the effects of cilostazol to the expression of LOX-1 mRNA and protein, superoxide generation and neointimal hyperplasia of the rat carotid artery after balloon injury. The injury was performed inserting the balloon catheter through the rat common carotid artery and after 14 days a histopathological analysis revealed a significant restenosis with smooth muscle cell proliferation and neointima formation that was associated with an enhanced expression of LOX-1, superoxide generation, Pretreatment of rats with cilostazol (100 mg/kg/day) reduced neointima formation, superoxide generation, and LOX-1 expression ($p<0.05$). Here, we show that Cilostazol reduces neointimal hyperplasia by inhibition superoxode generation and expression of lectin-like oxidised LDL receptor-1 after balloon common arterial injury in a rat model.

## e0211 SI MIAO YONG AN DECOCTION PROMOTES ATHEROSCLEROTIC PLAQUE STABILITY IN VULNERABLE PLAQUE RABBITS

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**Background** Si Miao Yong An decoction and its medicine Mailuoning injection are proved-effect medicine treating ischaemic cardiovascular disease, but whether it can stabilise the atherosclerotic plaque is currently no clear conclusions. We want to explore the role of Si Miao Yong An decoction interventing on rabbit aortic atherosclerotic plaque instability.
**Methods** Japanese white rabbits were divided into control group, model group, Simvastatin group and Si Miao Yong An group. The