Method The proliferative phase of VSMCs was evaluated by water-soluble tetrazolium (WST-1) assay. LTCC-α1C mRNA and protein were determined by reverse transcription-polymerase chain reaction (RT-PCR) and western blotting. Cell cycle, the ratio of G0 +G1 phase cells and S phase cells were analysed using flow cytometry analysis after VSMCs were exposed to fluvastatin.

Result Fluvastatin showed an inhibitory effect on VSMCs growth in a time-dependent manner as assessed by WST-1 assay, proliferation of VSMCs was suppressed by 2.1-fold after the administration of fluvastatin for 24 h. The down-regulation of LTCC-α1C subunit induced by platelet derived growth factor (PDGF)-BB was significantly restored by the treatment of fluvastatin. The mRNA and protein levels of LTCC-α1C subunit of cultured VSMCs were significantly decreased after incubation with PDGF (10 μg/l) ($p<0.05$). Incubation with fluvastatin (10^{-5} mol/l) induced a 4.1-fold increase in mRNA level and a 1.2-fold increase in protein expression of LTCC-α1C subunit. The ratio of G0 +G1 phase cells increased ($p<0.05$), and the percentage of S phase cells decreased ($p<0.05$), after VSMCs were exposed to fluvastatin for 24 h.

Conclusion Fluvastatin may induce cell growth arrest at G0, G1 phase of cell cycle and upregulate LTCC-α1C subunit expression in cultured VSMCs.

[gw22-e0073] **FLUVASTATIN UPREGULATES L-TYPE CA2+ CHANNEL A1C EXPRESSION AND INDUCES CELL ARREST IN VASCULAR SMOOTH MUSCLE CELLS**

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Objective To investigate the effect of fluvastatin on L-type calcium channel (LTCC) in the cultured aortic vascular smooth muscle cells (VSMCs) and its alteration associated with proliferation.