Abstract: **P165**

The dihydropyridine calcium antagonist nicardipine reduces aortic smooth muscle cell viability, proliferation and migration

**Authors:**
R Stamatiou¹, I Aidonidis¹, C Malavaki¹, E Paraskeva¹, A Hatziefthimiou¹, ¹University of Thessaly, Department of Medicine - Larissa - Greece,

**Topic(s):**
Vascular Biology and Physiology, Other

**Citation:**
Cardiovascular Research (2018) 114 (Supplement 1), S43

**Background/Introduction.** Hypertension is a common cardiovascular disease characterized by structural cardiac muscle and vascular smooth muscle cell (VSMC) hypertrophy, as a result of remodeling. The mechanisms that may initiate and maintain remodeling remain unsettled and need clarification in order to improve therapeutic strategies in clinical practice.

**Purpose.** To investigate the effect of nicardipine (NIC), a dihydropyridine calcium antagonist that is extensively used against hypertension, on the viability, proliferation and migration capability of aortic VSMCs. To test the hypothesis that NIC may exert additional effects against VSMC remodeling.

**Methods.** VSMCs were isolated from New Zealand rabbit aortic preparations. VSMCs were serum starved and then treated with 0.1-10 µ? NIC in the presence or absence of 10% FBS for 24-48h. Cell viability was assessed with cell count after Trypan-blue staining, while proliferation using the Cell Titer 96 AQueous One Solution Assay (MTS) method. VSMC morphology was observed with reverse microscopy and indirect immunofluorescence with anti-smooth muscle a-actin monoclonal antibody. The migration capability was estimated using the "wound healing" assay. All data are expressed as means±standard error of the mean and differences were analyzed by one way ANOVA with Bonferonni’s post hoc test or unpaired t-test with statistically significant differences being determined by Mann-Whitney test using GraphPad Prism 4.

**Results.** Treatment with NIC (0.1µ? -10µ?) reduced significantly cell viability and inhibited VSMCs proliferation in the presence of 10%FBS in a dose-dependent way, from 205.4±17.5% to 176.6±17%, 160.6±5.7%, 150.4±11.2%, 61.22±7.83% after 0.1µ?, 1µ?, 3µ?, 10µ? NIC treatment, respectively. Additionally, NIC affected the arrangement of smooth muscle a-actin and altered VSMC interactions. Furthermore, the treatment of VSMCs with NIC for 24h or 48h reduced significantly their migration, in a wound-healing assay, which was inhibited, from 41.7±1.3% to 15.4±0.5%, 7.5±1.7%, and from 85.2±5% to 26.9±2.8%, 19.3±2.5%, after 0.1µ? and 10µ? NIC treatment, respectively.

**Conclusion.** Treatment with NIC reduced viability and proliferation of VSMCs and inhibited their ability to migrate. These effects might suggest an anti-remodeling mechanism of NIC in addition to its anti-hypertension action.