2-P2-03

Poster Sessions

Effects of pressure stress on MAPK activation induced by interleukin-1 β and angiotensin II in cultured human pulmonary artery smooth muscle cells.

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Pulmonary artery smooth muscle cells (PASMCs) play an important role in a sequence of events leading to formation of pulmonary hypertension (PH). Nevertheless, little is known about the direct effects of high pressure on function and the intercellular signaling pathways of PASMCs. We recently showed that pressure stress delays a transient cyclooxygenase-2 expression by interleukin-1 β (IL-1 β) stimulation in cultured human PASMCs. The aim of this study was to evaluate the effect of pressure stress which simulates PH on IL-1 β or angiotensin II (Ang II)-induced MAPK, i.e., extracellular signal-regulated kinase (ERK), p38 MAPK, and c-jun N-terminal kinase (JNK), phosphorylations in cultured human PASMCs. Either 20 or 60 mmHg of an atmospheric pressure was given to PASMCs by a pressure-loading apparatus. Protein phosphorylation was analyzed by Western blotting. IL-1 β induced MAPK phosphorylations peaking at 5-15 min in non-pressurized cells, whereas these phosphorylations were delayed in the pressurized cells. Ang II induced ERK and p38 MAPK phosphorylations peaking at 15 min, but had no effect on JNK phosphorylation. Ang II-induced ERK and p38 MAPK phosphorylations were almost unaffected by the pressure stress. These results suggest that the effect of pressure stress on MAPK activation depends on regional bioactive substances.