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Origanum Syriacum Inhibits Proliferation, Migration, Invasion and Induces Differentiation of Human Aortic Smooth Muscle Cells

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Cardiovascular diseases (CVDs) are still the number one cause of morbidity and mortality both in Qatar and worldwide. A major risk factor of CVDs is atherosclerosis, the hardening of blood vessels caused by decreased diameter and formation of plaque. A key player in atherosclerosis prognosis is the switch of vascular smooth muscle cells (VSMCs) phenotype from their undifferentiated state to a synthetic one. The synthetic state of VSMCs is characterized by an increase in proliferation, migration and invasion to the lumen of blood vessels, contributing to the atherosclerotic plaque. Ineffectiveness of current treatments has lead to an increasing interest in herbal medicine, possibly because they are cheap and produce little side effects. *Origanum syriacum*, commonly known as Zataar, is an important constituent of the Mediterranean diet; a diet correlated with lower risk of CVDs. *O. syriacum* is also reported for its antioxidant and anti-inflammatory activities, an indication of its possible anti-atherosclerotic activities. However, *O. syriacum* effect on atherosclerosis or CVDs is not well studied. This is why we chose to study the effect of the ethanolic extract of *O. syriacum* (OSEE) on the proliferation, migration, invasion and differentiation of human aortic smooth muscle cells (HASMCs). Cell Titer-Glu assay was used to study OSEE effect on HASMCs viability. Cells were incubated with OSEE (0, 0.5, 0.1 and 0.2 mg/ml) for 24, 48 and 72 hours. OSEE has showed to exert a significant anti-proliferative effect on HASMCs. This effect, though, seems to be concentration-dependent, but not time-dependent. The optimum concentration, 0.2 mg/ml, significantly decreased HASMCs viability at 24 and 72 hours by 52.5 ± 10.39% and 47.6 ± 9.83% compared to control, respectively. A scratch-wound assay was used to determine OSEE's effect on migration of HASMCs. A monolayer of cells was scratched and wound size was measured every 2 hours for 24 hours. OSEE significantly inhibited the migratory capacity of HASMCs compared to untreated cells. Cells that were incubated with 0.2 mg/ml of OSEE for 24 hours showed 65.07 ± 12.58% less migration than the control. To measure the invasive capacity of HASMCs, Matrigel-coated BD BioCoatTM filter inserts were used. Cells were incubated in serum free media with or without 0.2 mg/ml of OSEE, and number of invasive cells was counted

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after 24 hours. OSEE has shown to significantly decrease the invasive capacity of HASMCs by 79.82 ± 5.69% compared to control. To study effect of OSEE on differentiation of HASMCs, western blotting was used to measure calponin-h1 activity. Cells were incubated with or without 0.2 mg/ml OSEE for 24 hours and lysate was analyzed. OSEE increased the expression of calponin-h1 by 147.19 ± 72.33% compared to control. These results indicate that OSEE possess anti-atherosclerotic abilities by modulating the phenotype of HASMCs. This modulation returns HASMCs to their differentiated state, as shown by calponin-h1 increase. It also exhibits this modulation by inhibition of the synthetic state phenotypes of proliferation, migration and invasion of HASMCs. This anti-atherosclerotic effect should be further studied by possibly investigating OSE's effect on specific pathways that leads to migration and invasion of HASMCs, such as ERK1/2 and MAPK pathways, as well as MMPs expression.