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INHIBITION OF SMOOTH MUSCLE CELL PROLIFERATION AND INTIMAL THICKENING WITH SMALL PEPTIDE MIMETICS OF SOLUBLE NCADHERIN

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Aim: We investigated whether a small peptide sequence from soluble Ncadherin (SNC) modulated intimal thickening by changing vascular smooth muscle cell (VSMC), fibroblast and endothelial cell (EC) behaviour. Restenosis of vein grafts is a significant problem resulting in failure in 30-50% of patients. Reduction of VSMC proliferation, without preventing re-endothelialisation is desirable. SNC reduced ex vivo intimal thickening and increased endothelial coverage. We wish to produce a smaller therapeutic peptide. **Methods:** Cells: Human saphenous vein VSMC and fibroblasts; HUVECs. **Proliferation:** EdU incorporation. **Migration:** scratch wound assay (distance migrated, mm). **Apoptosis:** cleaved caspase-3 immunocytochemistry. Human saphenous vein organ culture used as ex vivo model of intimal thickening, analysed by elastin van Gieson staining (intimal size), EdU (proliferation) and Q-Bend-10 (endothelial coverage). **Results:** The peptide significantly reduced VSMC proliferation ($14.4 \pm 4.8\%$ vs $3.58 \pm 2.2\%$, $n=4$, $p < 0.05$). **Conclusions:** A peptide mimetic of SNC is an attractive therapeutic for restenosis as it attenuates VSMC proliferation, without detrimental effects on re-endothelialisation. A peptide is inexpensive to produce and easy to deliver.

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