Molecular cloning and characterization of smLIM, a developmentally regulated LIM protein preferentially expressed in aortic smooth muscle cells.

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Abstract

Differentiated, quiescent vascular smooth muscle cells assume a dedifferentiated, proliferative phenotype in response to injury, one of the hallmarks of arteriosclerosis. Members of the LIM family of zinc-finger proteins are important in the differentiation of various cells including striated muscle. We describe here the molecular cloning and characterization of a developmentally regulated smooth muscle LIM protein, SmLIM, that is expressed preferentially in the rat aorta. This 194-amino acid protein has two LIM domains, and comparisons of rat SmLIM with its mouse and human homologues reveal high levels of amino acid sequence conservation (100 and 99%, respectively). SmLIM is a nuclear protein and maps to human chromosome 3. SmLIM mRNA expression was high in aorta but not in striated muscle and low in other smooth muscle tissues such as intestine and uterus. In contrast with arterial tissue, SmLIM mRNA was barely detectable in venous tissue. The presence of SmLIM expression within aortic smooth muscle cells was confirmed by in situ hybridization. In vitro, SmLIM mRNA levels decreased by 80% in response to platelet-derived growth factor-BB in rat aortic smooth muscle cells. In vivo, SmLIM mRNA decreased by 60% in response to vessel wall injury during periods of maximal smooth muscle cell proliferation. The down-regulation of SmLIM by phenotypic change in vascular smooth muscle cells suggests that it may be involved in their growth and differentiation.