Expression of the Novel Scavenger Receptor SR-PSOX in Cultured Aortic Smooth Muscle Cells and Umbilical Endothelial Cells

To the Editor:

In the November issue of *Arteriosclerosis, Thrombosis, and Vascular Biology*, Minami et al. demonstrated expression of the novel scavenger receptor for phosphatidylserine and oxidized lipoprotein (SR-PSOX) in lipid-laden macrophages accumulated in the intima of human atherosclerotic lesions. Because SR-PSOX seems to be identical to the membrane-anchored chemokine CXCL16, which may play a dual role in inflammation and homeostasis, Minami et al. discussed the potential regulation of SR-PSOX by pro-inflammatory cytokines. Although the authors did not detect SR-PSOX in smooth muscle cells (SMCs) and endothelial cells (ECs), they did discuss the possible expression of SR-PSOX in these cell types. Until now, only the expression of the scavenger receptors SR-AI/II, CD36, and LOX-1 has been described.

In our studies on the formation of SMC-derived foam cells during atherogenesis, we have focused on the expression of scavenger receptors, including SR-PSOX, in SMCs and ECs. We have also investigated the influence of cytokines on the expression of SR-PSOX in SMCs. Reverse transcriptase-polymerase chain reaction (PCR; primers for human SR-PSOX, 5'-TACAGGAGGTCCAGCTCCT-3' and 5'-GGGCGCTGTAGAACATTTA-3', porcine SR-PSOX, 5'-TATGTTCAAGGACGGACGGTAGC-3' and 5'-CTGCAAGGTGAATGAGCAGAT-3') was performed on total RNA from cultured human and porcine aortic SMCs and human umbilical vein endothelial cells (HUVECs). PCR was performed at 94°C (45 seconds), 58°C (60 seconds), and 72°C (60 seconds) for 20 to 40 cycles in the linear area of amplification. The sequences of the scavenger receptors SR-AI/II, CD36, and LOX-1 in SMCs have been described.

In summary, we demonstrate that the scavenger receptor SR-PSOX, which is expressed in human atherosclerotic lesions and may be involved in foam cell formation, is not only expressed in macrophages, but also in cultured SMCs and HUVECs. Moreover, our data indicate that SR-PSOX is governed by pathways other than those reported for SR-AI/II and LOX-1. Further studies will elucidate the functional role of SR-PSOX and its regulation in SMCs and ECs.

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In Response:

SR-PSOX/CXCL16 is expressed by macrophages, dendritic cells, and CD19+ B lymphocytes.1–3 Its expression can be upregulated, to some extent, by pro-inflammatory stimuli, such as bacterial endotoxin, tumor necrosis factor-α, and ligation to CD40.2,3 Currently, we are trying to elucidate whether other cell types, including vascular endothelial and smooth muscle cells, express SR-PSOX. Our preliminary data, so far, have shown that SR-PSOX mRNA expression is undetectable by northern blot analysis in cultured human vascular smooth muscle cells.4,5 LOX-1 in human smooth muscle cells, which is based on reverse transcriptase–polymerase chain reaction data shown in the letter by Hofnagel et al do not include appropriate negative controls, such as omission of reverse transcription. Therefore, contamination of genomic DNA in their RNA preparations might possibly affect their results. We do not agree with the conclusion by Hofnagel et al that SR-PSOX is more strongly expressed than LOX-1 in human smooth muscle cells, which is based on reverse transcriptase–polymerase chain reaction analysis alone. Further studies by use of neutralizing monoclonal antibodies and gene knockout mice would tell us more concerning the roles of SR-PSOX in macrophages and other cell types in a variety of pathophysiological settings including atherogenesis.

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