

# **SDF-1 $\alpha$ -induced dual pairs of E-selectin/ligand mediate endothelial progenitor cell homing to critical ischemia**

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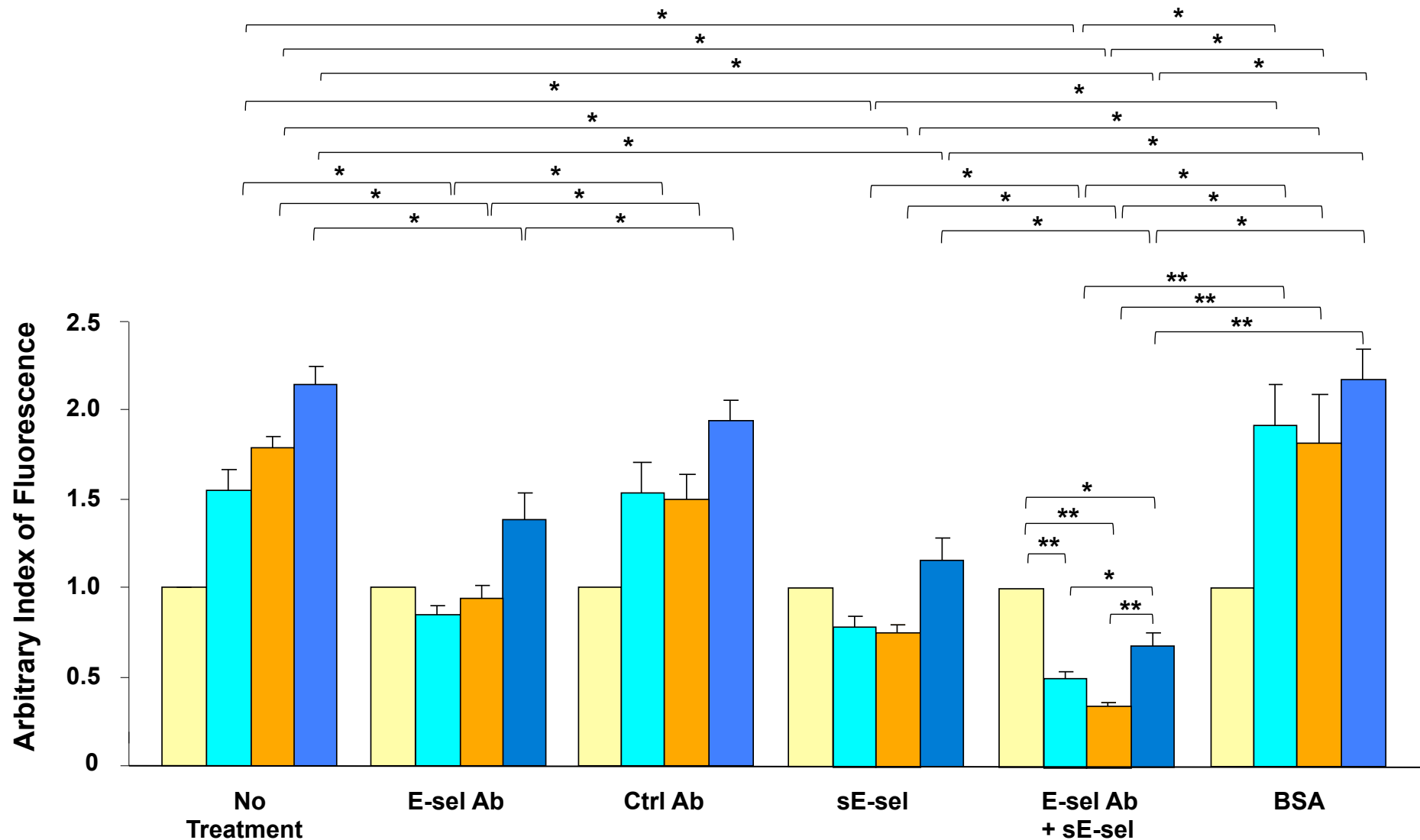
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# Supplemental Table I

## Fold changes of gene expression upon SDF-1 $\alpha$ stimulation in human EPC

<i>Gene name</i>		<i>Fold change</i>
Collagen, type XII, alpha 1	+	2.64
Collagen, type VII, alpha 1	+	3.12
Versican	+	3.61
Catenin	+	5.10
Integrin, alpha 2	+	2.27
Integrin, alpha 3	+	2.22
Integrin, alpha 4	+	3.71
Integrin, alpha V	+	3.71
Integrin, beta 1	+	5.66
Laminin, alpha 3	+	2.66
Laminin, beta 3	+	97.68
Laminin, gamma 1	+	2.04
Matrix metalloproteinase 1	+	3.63
Matrix metalloproteinase 10	+	3.14
Matrix metalloproteinase 12	+	8.82
Selectin E	+	3.48
Sarcoglycan, epsilon	+	2.36
Secreted phosphoprotein 1	+	5.17
Transforming growth factor, beta-induced, 68kDa	+	2.06
Thrombospondin 1	+	4.44
Thrombospondin 2	+	3.76
Tenascin C	+	3.34
ADAM metalloproteinase with thrombospondin type 1 motif, 1	-	2.51
Integrin, alpha 8	-	2.07
Matrix metalloproteinase 9	-	2.39

# Supplemental Figure 1



**SDF-1 $\alpha$ -induced E-selectin/ligands are responsible for mediating EC-EPC adhesion *in vitro*.** SDF-1 $\alpha$  increases adhesion of Dil-Ac-LDL<sup>+</sup> human EPC to the HMVEC monolayer. Single or combined treatment of SDF-1 $\alpha$ -stimulated HMVEC monolayers with sE-sel or treatment of SDF-1 $\alpha$ -stimulated Dil-Ac-LDL<sup>+</sup> EPC with E-selectin-neutralizing Ab can significantly inhibit EPC-HMVEC interaction. See Fig. 3 for the details of 4 combinations of EPC-HMVEC interactions and ANOVA test. \*  $P < 0.05$ ; \*\*  $P < 0.01$ .