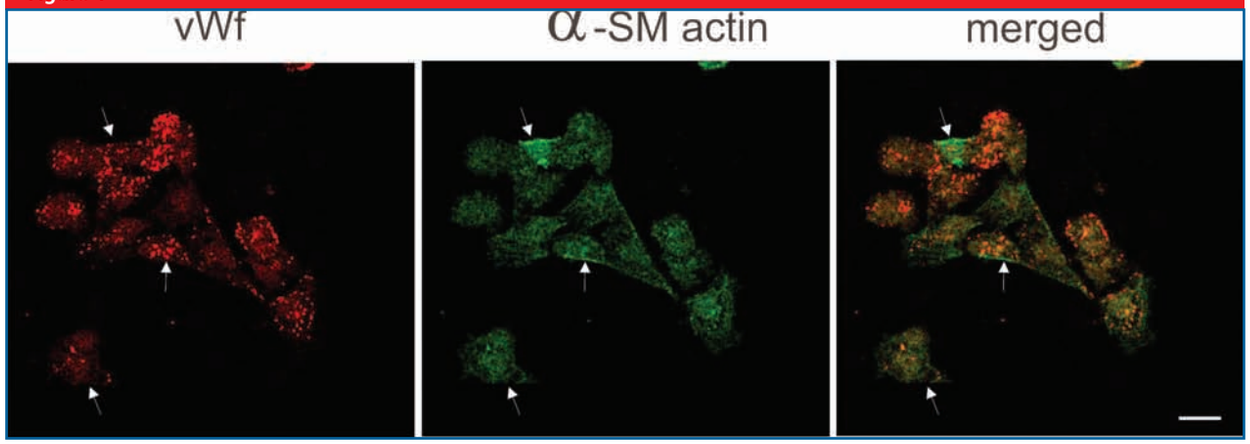
In addition to IGFII and EGF, the vascular endothelial growth factor (VEGF) and its receptor (VEGFR) has also been proposed to participate in vasculogenesis, angiogenesis, atherosclerosis, restenosis, and tumor progression[43-45], processes where both EMT and EndoMT are involved. For instance, VEGFR was found to form a complex with VE-cadherin, β-catenin in vascular endothelial cells enhancing VEGF signaling[46]. Induction of EMT by EGFR in tumor progression in pancreatic cancer has also been proposed[47]. However, studies focused on the role of VEGF/VEGFR signaling in the EndoMT process are scarce.

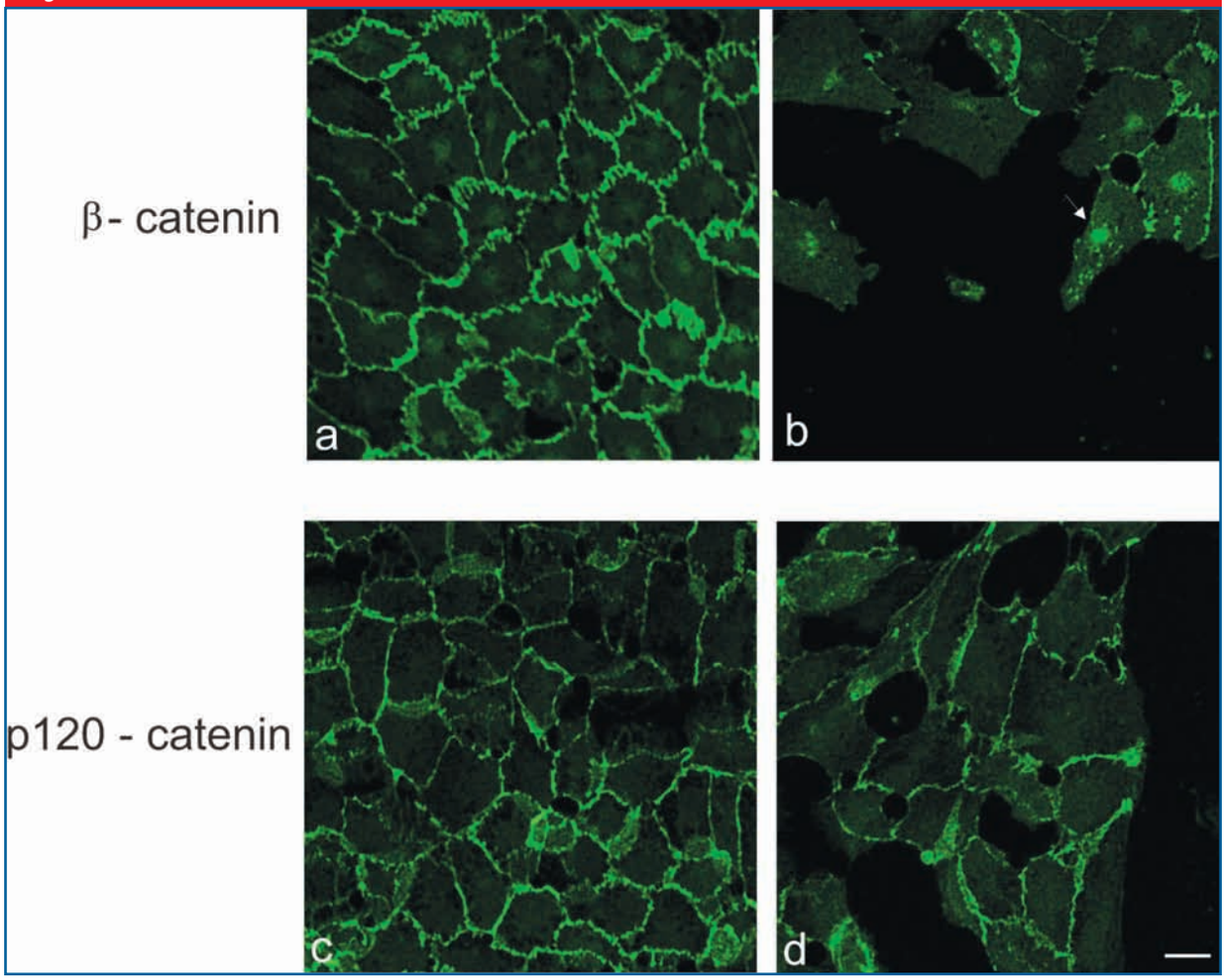


**Figure 1**




Confocal laser scanning microscope fluorescence (CLSM) images of vWf and α-SM actin in a monolayer of aortic embryonic endothelial cells attached to fibronectin after 48 h in culture in complete medium. Strong positivity for vWf (red) in punctated and granular pattern typical of endothelial cells, and for α-SM actin (green) delineating cellular margin are observed. Double immunofluorescence of the same field shows some of the cells exhibiting immunoreactivity for both vWf and α-SM actin (arrows, merged). Scale bar = 20μm.

**Figure 2**




CLSM fluorescence images of both β- and p120-catenins in a monolayer of embryonic endothelial cells attached to fibronectin after 22 h in culture.

Strong positivity for both β-catenin (a) and p-120 catenin (c) is observed at the plasma membrane of endothelial cells that display a cobblestone appearance, whereas this expression and distribution appeared altered in separating, detaching and migrating cells (b,d). Note that β-catenin immunoreactivity is also detected in the nucleus and cytoplasm of some migrating cells (arrow).