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e endothelial-to-mesenchymal  
transition in angiogenic endothelial  
cells associated with the  
antiangiogenic actions of S100A4.  
S100A4 has recently been  
demonstrated to be an inhibitory  
mediator of angiogenesis. This study  
therefore investigated the effects of  
S100A4 on the regulation of capillary  
tube formation in vitro and on the  
vascular network formation in vivo.  
In addition, the underlying molecular  
mechanism of S100A4-mediated  
antiangiogenesis was studied. In  
vitro, S100A4 inhibited human  
umbilical vein endothelial cell  
(HUVEC) tubulogenesis on Matrigel  
at 20 ng/ml. In contrast, at lower

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S100A4 concentrations, platelet-derived growth factor-BB (PDGF-BB)-stimulated HUVEC produced significantly more tubules than control HUVEC. In the zebrafish assay, the inhibition of angiogenesis by S100A4 was indicated by a reduction in the number and length of intersegmental vessels. Inhibition of capillary tube formation by S100A4 was mediated through the proteolytic cleavage of PDGFR-beta and activation of its downstream signaling pathway. Moreover, S100A4 inhibited the proliferation of HUVEC and arrested HUVEC in the G(0)-G(1) phase. We also found that S100A4 overexpression decreased the expression of VE-cadherin and increased the expression of

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mesenchymal cell markers in vitro. In vivo, angiogenesis was significantly impaired in an in vivo Matrigel plug assay in S100A4-overexpressing fibroblasts and in S100A4-knockdown HUVEC (in vitro). These findings suggest that S100A4 is an important regulator of angiogenesis through the inhibition of endothelial cell migration and proliferation, and that S100A4 suppresses angiogenesis through the induction of an endothelial-to-mesenchymal transition phenotype. Mannen, som er dømt for vold mot en kvinne på en jernbanestasjon i 2018 og utenfor rettssalen, skal tilbakeholde seg fra politiet. 5ec8ef588b

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