Pin1 as a Protector of Vascular Endothelial Homeostasis

To the Editor:

In their recently published article in Hypertension, Chiasson and colleagues,1 in contrast to Ruan et al.2 demonstrated that binding of Pin1 to endothelial NO synthase (eNOS) facilitates the dephosphorylation and activation of the enzyme. Thus, the activated eNOS increases NO production, leading to endothelium-dependent dilation and blood pressure regulation.1 Although there seems an implication that Pin1 directly results in serine-116 dephosphorylation of eNOS, they clearly showed that Pin1 deficiency negatively affects eNOS-regulated endothelial functions. However, in an editorial commentary for this study published in the same issue,3 the authors consider Pin1 as a phosphatase. In addition, they accentuate this mistake clearly in their Figure. Furthermore, they state that the findings of this study are consistent with the previous study done by Ruan et al.,2 on their Figure. Additionally, they demonstrate how Pin1 facilitates dephosphorylation of eNOS at serine-116 by inducing the activation of calcineurin, a Ca2+/calmodulin-dependent protein phosphatase (PP2B).5 On the other hand, Pin1 induces transcriptional upregulation of vascular endothelial growth factor.6 Taken together, Pin1-mediated increased activation of VEGF may result in dephosphorylation and activation of eNOS at serine-116. Thus, whereas Pin1 deficiency negatively influences endothelial function as demonstrated by Chiasson et al.,1 deficiency of this important isomerase could also lead to the attenuation of VEGF-mediated angiogenesis (Figure).

Disclosures

None.

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References


Figure. Pin1 upregulates the expression of vascular endothelial growth factor (VEGF) that, in turn, activates the phosphatase PP2B. Increased VEGF influence in the vessel wall and dephosphorylation of endothelial NO synthase (eNOS) at serine-116 (S116) by PP2B activates eNOS toward the protection of endothelial homeostasis.
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