Plasminogen Activator Inhibitor Type-2 (PAI-2)

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### Plasminogen Activator Inhibitor Type-2 (PAI-2)  
**Latest scientific evidence**

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**Summary:**  
Plasminogen activator inhibitor (PAI)-2 is a determinant of placental development, besides fms-like tyrosine kinase (sFlt)-1, placental growth factor (PIGF) and the B vitamin folate. In their study Bouwland-Both and colleagues analysed the association between these maternal determinants and early fetal size. The four biomarkers including PAI-2 were found to be positively correlated with first-trimester fetal size. |
**Summary:**  
In this study Zhao and colleagues used PAI-2 (serpinB2) deficient mice to analyse the host response to the enteric nematode, *Heligmosomoides bakeri*. Nematode infections in mice result in increased intestinal PAI-2 expression. In PAI-2 deficient mice this up-regulation together with concomitant IL-4 and IL-13 expression was attenuated coincident with an impaired worm clearance. Also the nematode-induced smooth muscle hypercontractability was lost and the infection-induced increase in mucosal permeability was delayed. In isolated macrophages CCL2 expression was reduced. The authors conclude that “immune regulation of serpin B2 expression plays a critical role in the development of Th2-mediated protective immunity against nematode infection by a mechanism involving CCL2 production and macrophage infiltration”. |
**Summary:**  
PAI-2 lacks an N-terminal signal peptide and upon stimulation accumulates in cells and only a small percentage of it is secreted. However, under inflammatory conditions the extracellular concentration of PAI-2 increases. Boncela and colleagues explored the possible mechanism that may underlie the secretion of PAI-2 from endothelial cells stimulated with lipopolysaccharide (LPS). They found that stimulation generated PAI-2 containing structures that resemble secretory vesicles. “These vesicles may represent the mechanism by which high local concentrations of serpinB2 are released at inflammation sites from endothelial cells.” |
Plasminogen Activator Inhibitor-2 Polymorphism Associates with Recurrent Coronary Event Risk in Patients with High HDL and C-Reactive Protein Levels.


**Summary:**
Corsetti and colleagues analysed whether the PAI-2 polymorphism rs6095 is associated with recurrent cardiac events. Indeed, Kaplan Meier analysis revealed that carriers of the variant allele had a much higher risk of recurrent coronary events. The authors interpret this finding in view of the possible role of PAI-2 in inhibition of plasminogen activators especially in inflammatory foci.

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TANK-binding kinase 1 (TBK1) controls cell survival through PAI-2/serpinB2 and transglutaminase 2.


**Summary:**
Delhase and colleagues addressed the fact that genetic studies in mice have identified the TANK-binding kinase 1 (TBK1) as a regulatory molecule that promotes survival downstream of Tumor Necrosis Factor signalling. The mechanism by which TBK1 exerts its survival function remained elusive. In a mouse model they provided evidence for “PAI-2 and transglutaminase 2 as downstream mediators in the anti-apoptotic response triggered upon TBK1 activation.”

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Association of plasminogen activator inhibitor type 2 (PAI-2) with proteasome within endothelial cells activated with inflammatory stimuli.


**Summary:**
PAI-2 synthesis can be rapidly stimulated in endothelial cells by inflammatory stimuli. Boncela and colleagues analysed the interaction between PAI-2 and proteasomes in cultured endothelial cells. By transfection experiments together with co-immunoprecipitation as well as electron and confocal microscopy, they provide evidence that PAI-2 interacts as an inhibitor with proteasomes. Taken together, they suggest “that PAI-2 in endothelial cells induced with inflammatory stimuli, can inhibit proteasome and thus tilt the balance favouring proapoptotic signalling”.

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