P-selectin translocation is mediated by angiopoietins in bovine aortic endothelial cells (BAEC)

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Key words: Angiopoietins, P-selectin translocation, endothelial cells, confocal microscopy, deconvolution, volume rendering.

Recently identified, angiopoietin-1 (Ang1) and -2 (Ang2) bind to the tyrosine kinase receptor Tie2 and contribute to orchestrate blood vessel formation during angiogenesis [1]. Ang1 mediates vessel maturation and integrity by favouring the recruitment of pericytes and smooth muscle cells. Ang2, initially identified as a Tie2 antagonist may, under certain circumstances, induce Tie2 phosphorylation and biological activities. Since inflammation exists in a mutually-dependent association with angiogenesis, we sought to determine if Ang1 and/or Ang2 could modulate proinflammatory activities, namely P-selectin translocation, in bovine aortic endothelial cells (BAEC) and dissect the mechanisms implicated. P-selectin, an adhesion molecule found in Weibel-Palade bodies (WPB) of endothelial cells (EC), is rapidly translocated to the cell surface upon EC activation during inflammatory processes [2]. Herein, we report that Ang1 and Ang2 (10^{-9} M) are both capable of mediating a rapid and transient endothelial P-selectin translocation at the plasma membrane as assessed by confocal microscopy, deconvolution and volume rendering.

References: