Summer 2013

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**Recommended Citation**

Perez, Blanca; Brewster, Jay L.; and Jasperse, Jeffrey, "Effect of Shear Stress on ecNOS Expression and Dilation in Soleus Feed Arteries" (2013). Pepperdine University, *Featured Research*. Paper 57.  
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Effect of Shear Stress on ecNOS Expression and Dilation in Soleus Feed Arteries
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Abstract
Shear stress causes artery dilation and increased expression of endothelial cell nitric oxide synthase (ecNOS) in coronary and placental arteries. We sought to determine the importance of shear stress in maintaining normal dilation and normal levels of ecNOS in rat soleus feed arteries (SFA). SFA were isolated from male Sprague-Dawley rats and cannulated for in vitro microscopy (Fig. 6). SFA were exposed to no shear stress, low shear stress, or high shear stress conditions for 4 hours. After 4 hours, endothelium-dependent dilation (acetylcholine: Ach) and endothelium-independent dilatation (sodium nitroprusside: SNP) were tested. Arteries were then unclutched, mRNA was isolated, and RT-PCR for ecNOS mRNA was performed to determine whether shear stress altered ecNOS gene expression. Shear stress did not alter dilation to Ach, but dilation to SNP was greater in the high shear stress arteries. ecNOS mRNA content was greater in high shear stress arteries than low shear stress arteries. These data indicate that altered wall shear stress conditions alter ecNOS gene expression and vascular smooth muscle function.

Hypotheses
We hypothesized the following:
• Endothelium dependent dilation will be reduced by a period of reduced shear stress
• Endothelium independent dilation will not be altered by a period of reduced shear stress
• ecNOS gene expression will be reduced by a period of reduced shear stress

Introduction
Control of blood flow is accomplished by regulating the diameter of the arteries and arterioles feeding different organs. Various neural, hormonal, chemical and mechanical mechanisms contribute to the constriction and dilation of arteries. Shear stress, the frictional force between the blood and the endothelial cells, is one of these mechanical mechanisms. Shear stress has been found to have immediate effects, such as endothelial cell secretion of nitric oxide (NO), and long term effects, such as increased expression of endothelial cell nitric oxide synthase (ecNOS) in coronary arteries and placental arteries (2,3). However, the role of shear stress in ecNOS expression in skeletal muscle arteries is unknown.

Methods
• Soleus feed arteries were isolated from male Sprague-Dawley rats.
• Artery average maximal diameter was 200.6 ± 7.9 um.
• Feed arteries developed average spontaneous tone of 42.2 ± 3.4%.
• Artery diameter was measured using video microscopy (Fig.6)
• High, low, and no flow were induced in individual arteries for 4 hrs to create shear stress.
  • High flow = 200 uL/min
  • High shear stress = 135 dynes/cm²
  • Low flow = 5 uL/min
  • Low shear stress = 25 dynes/cm²
• Dose response curves in half log increments (10⁻³ M to 10⁻¹ M) of the following drugs were performed:
  • Acetylcholine (ACH): endothelium dependent dilator
  • Sodium nitroprusside (SNP): endothelium independent dilator
• mRNA was isolated from individual soleus feed arteries
• RT-PCR of mRNA from individual soleus feed arteries was used to measure ecNOS expression (Fig. 5).

Results

Figure 1: Soleus feed arteries under different shear stress conditions all responded similarly to acetylcholine (endothelium dependent dilator).

Figure 2: Endothelium independent dilation is reduced by decreased shear stress. (*) High shear stress different than low shear stress (P<0.05)

Figure 3: Soleus feed arteries under high shear stress demonstrate an increase in ecNOS mRNA expression

Figure 4: Soleus feed arteries under high shear stress demonstrate increased ecNOS mRNA expression (RT-PCR runs 1,3,4)

Conclusions
In Soleus Feed Arteries:
1. Low shear stress (reduced flow) did not alter endothelium dependent dilation (Fig.1).
2. Low shear stress (reduced flow) reduced endothelium independent dilation (Fig. 2).
3. High shear stress (high flow) increased the expression of ecNOS mRNA relative to no flow (Fig. 3).

Acknowledgements
• Funding provided by Pepperdine University and the NSF
• I would like to thank my PI Dr. Jasperse for his guidance in my research, Dr. Jay Brewster for his help in my RT-PCR method, and thank you to Samara Jasperse and Tanner Hecke for their technical assistance in my experiments

References