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Data Article

Diabetes alters vascular mechanotransduction data: Pressure-induced regulation of Nf-kapa-B p65 and translational associated signaling in the rat inferior vena cava

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ABSTRACT

Diabetic patients have a high rate of vein graft failure due to attrition or vessel occlusion that cause recurrent ischemic events or vein graft. Veins grafted into a high-pressure arterial environment must undergo vascular remodeling to better handle the altered hemodynamics and intravascular increased pressure. Multiple cellular and molecular events are purported to be associated with vascular remodeling of veins. Understanding the effect diabetes has on vascular mechano-transductive response is critical to decreasing graft failure rates. This article represents data regarding a study published in Cardiovascular Diabetology [1] and Open Journal of Endocrine and Metabolic Diseases [2] with the purpose of evaluating the effect of pressurization on rat inferior venae cavae (IVC). Here we provide the information about the

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method and processing of raw data related to our prior publish work and Data in Brief articles [3,4]. The data contained in this article evaluates the contribution of NF-kB signaling and associated proteins. IVC from lean and obese animals were exposed to a 30 min of perfusion at 120 mm Hg pressure and evaluated for changes in expression and (IkB-alpha, NF-kB p50, NF-kB p105, NF-kB p65, Traf2, caspase 12), phosphorylation of (IkB-alpha (ser 32), Fox01 (ser 256), and Fox04 (ser 193)) proteins thought to be involved in the regulation of vascular mechanotransduction.

Specifications Table

Subject area	*Biology*
More specific subject area	*Cardiovascular diabetic surgical tissue response*
Type of data	*graph, figure*
How data was acquired	*immunoblotting*
Data format	*analyzed*
Experimental factors	*IVC mounted vessels were subjected to 120 mm Hg of pressure for 30 minutes. Protein was then isolated from tissue for western blot analysis.*
Experimental features	*IVC obtained from Lean and Obese male Zucker rats were used in this experiment*
Data source location	*Huntington, WV USA*
Data accessibility	*Data is with this article and is related to articles published and in review* [1–4]

Value of the data

- The data presented in this Brief is vital to understanding the effect of diabetes on venous mechanotransduction.
- This data gives insight into the how diabetes alters tissue response to stimuli.
- This data provides a more thorough understanding of the NF-kB involvement in pressure mediated signaling in both diabetic and non-diabetic venous tissue.

1. Data

1.1. NF-kB p50 and p105

To determine the effect of pressurization of inferior vena cava (IVC) from diabetic male obese syndrome-X Zucker (OSXZ) diabetic and nondiabetic male normal lean Zucker (LNZ) animals we evaluated the expression of nuclear factor kappa-light-chain-enhancer of activated B cells (NF-kB p50 (NFkB2) [5,6]. IVCs obtained from the OSXZ control group showed no significant difference in the expression of NF-kB p105 when compared to the LNZ control animals. Pressurization resulted in a significant increase in NF-kB p105 in the LNZ IVC (148 $\pm$ 9.3%, $p < 0.05$) but did not illicit an increase in the levels of NF-kB p105 in the OSXZ IVC (Fig. 1-A). Compared to LNZ controls, NF-kB p50 was elevated in the OSXZ control IVC (74 $\pm$ 7.1%, $p < 0.05$). Pressurization of the IVC resulted in a