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Data Article

Perivascular adipose tissue mediated aortic reactivity data: Female lean and obese Zucker rats

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ABSTRACT

Perivascular adipose tissue (PVAT) is recognized as a paracrine organ that controls vascular function. One of the early data demonstrated PVAT from male Sprague-Dawley rats altered aortic vascular reactivity [1]. Subsequent studies have suggested PVAT mediated vascular reactivity is impaired in a variety of vascular beds with animal models of metabolic syndrome [2]. Findings in these experimental animals are generally reported by only male data. Here we report the new data on the effects of PVAT on the aortic reactivity of female lean zucker rats (LZR) and obese zucker rats (OZR). The data presented here is related to a recent manuscript entitled "Aortic dysfunction in metabolic syndrome mediated by perivascular adipose tissue TNFα- and NOX2-dependent pathway" [3] which demonstrated PVAT from male obese Zucker rats (OZR) impaired endothelial function of aorta which is associated with altered PVAT inflammatory signaling.

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Specifications Table

Subject	Physiology
Specific subject area	Metabolic syndrome, vascular pharmacology
Type of data	Graph Figure
How data were acquired	Myograph (AD Instrument DMT 620 M) Immunoblot and western blot imaging (Syngene G: BOX XT4)
Data format	Raw and analyzed
Parameters for data collection	Female 17-wk-old lean and obese Zucker rats (LZR and OZR, respectively) were used to collect aortic reactivity data with and without perivascular adipose tissue (PVAT) exudate. The biochemical data were collected from PVAT in female LZR and OZR.
Description of data collection	Isometric tension studies were performed on aortic rings from LZR and OZR. Vascular reactivity was assessed with and without PVAT conditioned media. Western Blot analyses were performed on whole tissue lysates of PVAT using the standard Western blot technique.
Data source location	West Virginia School of Osteopathic Medicine, Lewisburg, WV West Virginia University, Morgantown, WV
Data accessibility	The raw data files are provided with the article as supplementary files
Related research article	E. DeVallance, K.W. Branyan, K. Lemaster, I.M. Olfert, D.M. Smith, E.E. Pistilli, J.C. Frisbee, P.D. Chantler, Aortic dysfunction in metabolic syndrome mediated by perivascular adipose tissue TNFα- and NOX2-dependent pathway., Exp. Physiol. 103 (2018) 590–603. https://doi.org/10.1113/EP086818.

Value of the Data

- Vascular reactivity data from female LZR and OZR are scarce and these data from female metabolic syndrome rats can be used as a reference regarding the severity of impaired aortic reactivity.
- Lack of PVAT mediated vascular dysfunction from female LZR and OZR aortic reactivity data are interesting as opposed to the PVAT mediated vascular dysfunction male LZR and OZR aortic reactivity data [3].
- The PVAT biochemical data are also valuable for elucidating the mechanisms of vascular effects in response to PVAT incubation in female OZR as opposed to male OZR data [3].

1. Data description

Compared to the female LZR, body mass was 133% higher in female OZR ($n = 4$ each group; LZR 225.3 ± 25.9 vs OZR 524.3 ± 39.6 g; $p < 0.05$, Fig. 1 A). In a previous study [3], aortic vascular reactivity to an incremental dose of methacholine (Mch), a vascular endothelium dependent agonist, was significantly impaired in 17wks-old male OZR compared with age matched male LZR. Similarly, endothelium dependent dilation (EDD) assessed by Mch dose response was significantly impaired in 17 wks-old female OZR ($n = 4$ each group; $p < 0.05$, Fig. 1 B, LZR vs OZR). Inhibition of nitric oxide (NO) production with NG-nitro-L-arginine methyl ester (L-NAME) abolished metabolic syndrome associated differences in female Zucker rats (Fig. 1 B). The levels of maximum Mch induced dilation from aortic rings were 90.3 ± 6.1 and 73.7 ± 11.1% in LZR and OZR, respectively ($n = 4$ each group; $p < 0.05$, Fig. 1C). In contrast, endothelium independent dilation assessed by sodium nitroprusside (SNP) dose response were similar in the 2 groups. ($n = 4$ each group; $p =$ n.s., Fig. 1C, Max SNP dilation: 109.0 ± 9.1 vs 101.3 ± 3.3 for LZR and OZR).

White adipose tissues (WAT) and brown adipose tissues (BAT) have been recognized as major types of fat tissues with distinctive phenotypic expression patterns. PVAT around the thoracic aorta has morphological characteristics and protein expression features similar to BAT, such as high expression of UCP1 [4]. Our data showed altered PVAT phenotype in female OZR (Fig. 2 A) and UCP1 expression was decreased by 50.4% compared with the LZR PVAT ($n = 4$ each group; $p < 0.05$, Fig. 2 B 1.0 ± 0.1 vs 0.5 ± 0.2). This is consistent with a previous study using male LZR and OZR has demonstrated that UCP1 expression was significantly lower in PVAT from male OZR [3]. To determine the inflammatory signaling in PVAT from LZR and OZR, we assessed the expression and phosphorylation of STAT3 from the total tissue lysates. Immunoblot data from the female OZR PVAT showed phosphorylation of STAT3 increased expression by almost 5-fold when compared to the female LZR PVAT ($n = 4$ each group; $p < 0.05$, Fig. 2C 1.0 ± 0.4 vs 5.4 ± 2.6).

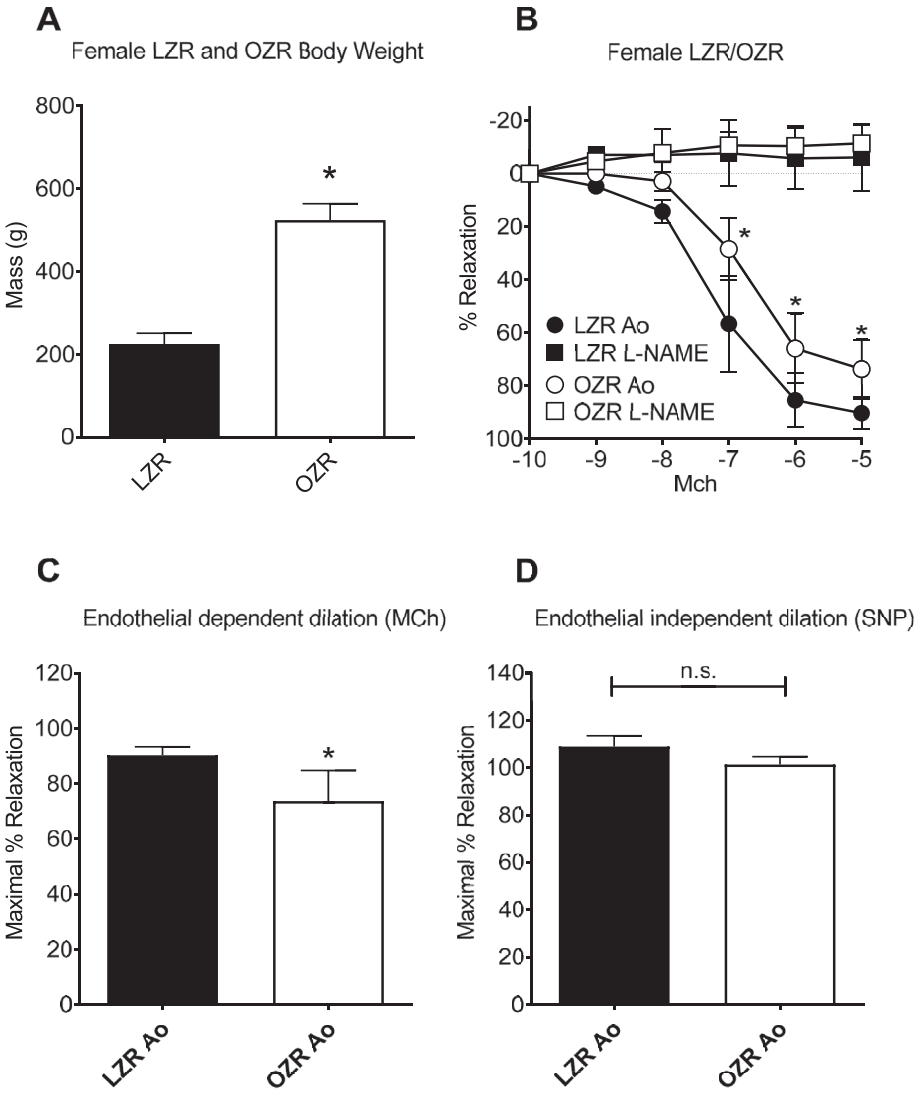

Fig. 1. Impaired endothelial dependent dilation in female OZR. (A) Body mass data from female lean and obese Zucker rat (LZR and OZR) in this study. Data are presented as mean ± standard deviation (SD, n = 4). (B) Relaxation of *in vitro* aortic rings from female LZR and OZR in response to increasing concentrations of methacholine (Mch). Data (Mean ± SD, n = 4) are presented for aorta from female LZR and OZR under control conditions and after pretreatment of the tissue with nitro-L-arginine methyl ester (L-NAME, square symbol). Asterisks indicate $p < 0.05$ vs. LZR by two-way repeated ANOVA (Mean ± SD, n = 4). (C & D) Group data of maximal endothelial dependent relaxation and endothelial independent relaxation. Data are presented as mean ± standard deviation (SD, n = 4). Asterisks indicate $p < 0.05$ vs. LZR by *t*-test (Fig. 1 A & C) and two way ANOVA (Fig. 1 B, Mean ± SD, n = 4).

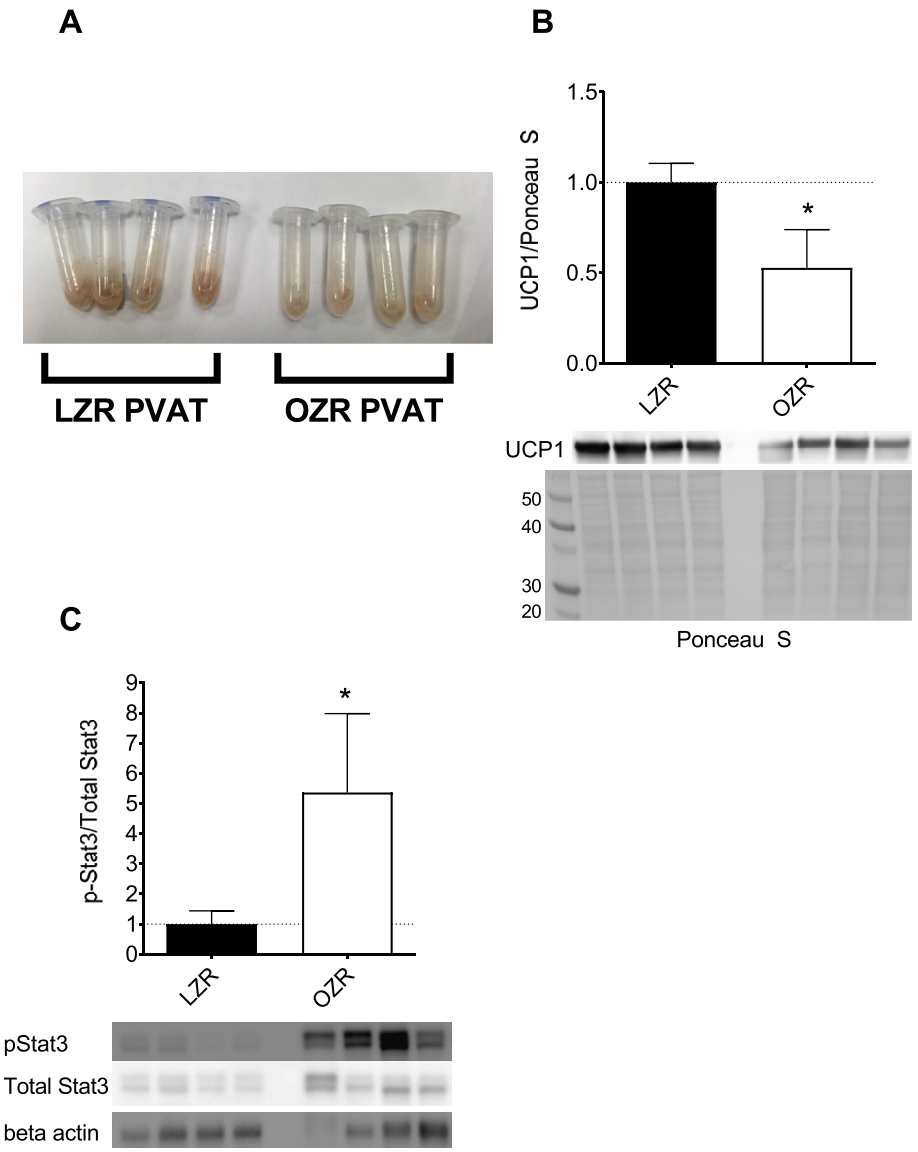

Fig. 2. PVAT phenotype and indices of inflammation. (A) An image of isolated perivascular adipose tissue (PVAT) from female LZR and OZR. (B) Relative expression levels of UCP1 in whole tissue lysate of PVAT from female LZR and OZR. (C) Total and phosphor STAT 3 in PVAT from female LZR and OZR. Data are normalized to mean of LZR PVAT and bands are shown below graphs. Asterisks indicate $p < 0.05$ vs. LZR by *t*-test (Mean ± SD, n = 4).

A recent study demonstrated that aortic rings from both male LZR and OZR incubated with their PVAT conditioned media (PVAT CM) had significantly impaired EDD, although the aortic reactivity was better maintained in male LZR incubated with LZR PVAT CM [3]. However, our data showed that EDD was not different in the female LZR aortic rings incubated with LZR PVAT CM compared with LZR aortic ring without PVAT CM. Similarly, EDD from female OZR aortic rings incubated with OZR PVAT CM were not impaired. (Fig. 3 A). The levels of maximum Mch induced dilation from aortic rings were 93.5 ± 4.9, 93.7 ± 4.1, 74.3 ± 11.7 and 83.3 ± 10.4% in LZR aorta, LZR aorta with PVAT CM, OZR aorta and OZR aorta

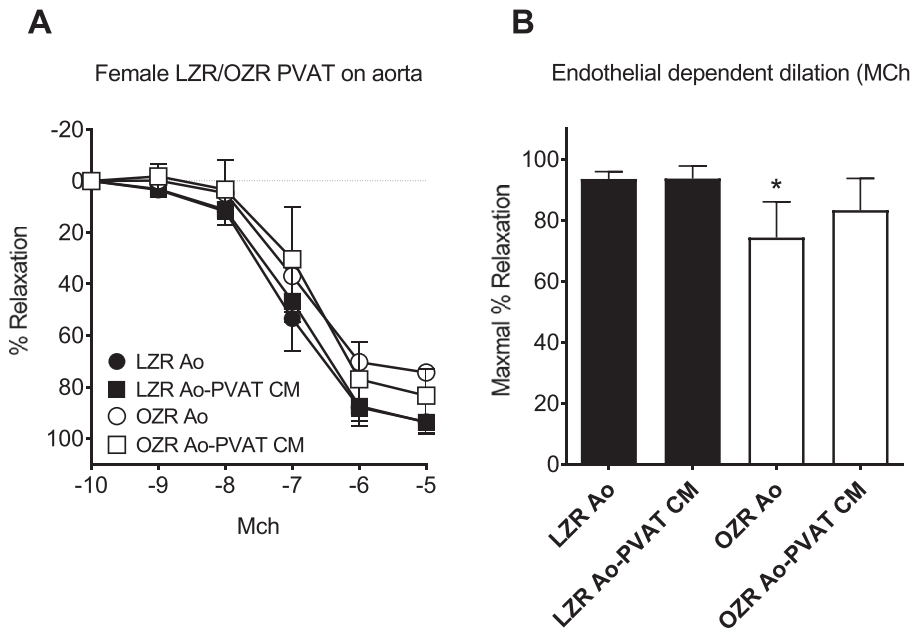

Fig. 3. Effect of PVAT on aortic endothelial dependent dilation. Perivascular adipose tissue (PVAT) has no effect on endothelial dependent dilation in both female LZR and OZR aortic rings. (A) Relaxation of *in vitro* aortic rings from female LZR and OZR in response to increasing concentrations of methacholine (Mch) with and without PVAT-conditioning media (PVAT-CM). (B) Group data of maximal endothelial dependent relaxation from female LZR and OZR aorta with and without PVAT-CM treatment. Data are presented as mean ± standard deviation (SD, n = 4). Asterisks indicate $p < 0.05$ vs. LZER by one way ANOVA (Mean ± SD, n = 4).

with PVAT CM, respectively ($n = 4$ each group; $p < 0.05$, Fig. 3 B), even thought our experimental approach was identical to the previous study [3].

2. Experimental design, materials, and methods

2.1. Animals

Animal ordering and housing conditions were similar to the previous study [3]. Briefly, female lean Zucker (LZR, n = 4) and obese (OZR, n = 4) rats were purchased at 8 wks of age from Envigo Laboratories and housed in the AAALAC approved animal care facility at the West Virginia University Health Science Center. Food and water were provided to animal ad libitum. Rats were euthanized at 17 wks of age and the all experiments described below were completed. All animal procedures were approved by the West Virginia University Institutional Animal Care and Use Committee and followed the guidelines in the Guide for the Care and Use of Laboratory Animals (National Academy Press, 2011).

2.2. Material

Unless otherwise stated in the methods, all chemicals used in organ bath experiments were purchased from Sigma. Antibodies against phosphor-Stat3 (# 9145), Stat3 (# 9139) and beta actin (# 8457), were purchased from Cell Signaling Technology. The antibody against UCP1 (Ab23840) was purchased from Abcam.

2.3. Vascular reactivity

Isometric tension experiments were performed as previously described [5,6]. After euthanizing the animals, the aorta was excised and then perivascular fat was carefully removed in ice cold

HEPES buffered physiological saline solution containing (mM) 135 NaCl, 5 KCl, 2 $CaCl_2$, 1 $MgCl_2$, 10 glucose, 10 HEPES and 5 Tris; pH 7.4. Four aortic rings (2−3 mm wide lengths) were cut from the aorta and mounted on the multi-channel organ bath system (DMT 620 M). The bath solution was bicarbonate buffered physiological saline solution (PSS) containing (mM) 130 NaCl, 4.7 KCl, 1.18 KH_2PO_4, 1.17 $MgSO_4$, 14.9 $NaHCO_3$, 5.5 Glucose, 0.026 EDTA, and 1.6 $CaCl_2$ aerated with 95% O_2 plus 5% CO_2 to maintain pH 7.4. The servo control system maintained buffer temperature at 37 °C throughout the experiments. The buffers were changed every 30 min during the experiments, and 3−5 3-5 washes were performed between the dose response experiments. Isometric tension was continuously recorded using a force displacement transducer (DMT 620 M) connected to an amplifier (PowerLab, AD Instruments) and the data were digitized at 100 Hz using a LabChart (AD Instruments).

Before starting the dose response experiments, aortic rings were stretched to reach an optimal tension that was determined according to the DMT's protocol. The vessels were equilibrated for 1 hour, and then, vessel viability was determined by KCL induced constriction (iso-potassium PSS: 60 mM KCl equimolar NaCl replacement of PSS). After the wash, endothelial function was assessed with methacholine dose response experiments (Mch:1×10^{-9} to 1×10^{-5} mol/L) after phenylephrine (PE: 10^{-6} M) induced contraction. Relaxation was calculated as the percentage of relaxation for each dose of Mch by the following equation [3]:

$$\text{Relaxation } (\%) = (z - x) / (z - y) \times 100$$

where z is the tension after PE: 10^{-6} M, x is the tension after a given dose of MCh, and y is the baseline tension. For the nitric oxide component of endothelial dependent dilation, Mch dose response was repeated following a 30-min incubation in the presence of the NG-nitro-L-arginine methyl ester (L-NAME: 10^{-4} M). For the effects of PVAT on endothelial function, PVAT conditioned media (PVAT CM, 200 mg/mL) was prepared from freshly isolated PVAT in HEPES buffered physiological saline solution for 2 hrs at 37 °C as described previously [3,7]. Fifty ul of PVAT CM was added to the bath containing 5 mL of PSS for 30 min. After the incubation, relaxation curves were performed as described above. Similarly, vascular smooth muscle reactivity was determined by sodium nitroprusside (SNP: 1×10^{-9} to 1×10^{-5} mol/L) after phenylephrine (PE: 10^{-6} M) induced contraction.

2.4. Immunoblotting

Semiquantitiative immunodetection of specific proteins was performed using established methods as described previously [8]. In brief, PVAT were homogenized in RIPA buffer with protease and a phosphatase inhibitor (Sigma). Samples were sonicated on ice, then incubated for 15 min on ice, and centrifuged at 10,000 g at 4 °C for 10 min. The supernatant was removed and protein concentrations were determined by BCA method using BSA as a standard (Pierce). Samples were diluted in Laemmli sample buffer and boiled at 95 °C for 5 min. Equivalent amounts of protein were separated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE), followed by a transfer onto nitrocellulose membranes. The membranes were stained with Ponceau S (Sigma) to verify successful transfer steps. For immune-detection, membranes were blocked with 5% nonfat milk in Tris-buffered saline-Tween (TBST) for 1 h at room temperature, washed 3 times in TBST, and incubated overnight at 4 °C in primary antibodies against Phosphor-Stat3 (Cell Signaling Technology # 9145, 1:500 dilution), Stat3 (Cell Signaling Technology # 9139, 1:500), beta actin (Cell Signaling Technology # 8457, 1:2000) and UCP1 (Abcam, ab23840, 1:500). After the overnight primary antibody incubation, the membranes were washed 3 times in TBST and incubated with anti-species secondary antibody conjugated with HRP (1:5000; Cell Signaling Technology) for 1 h at room temperature. SuperSignal™ West Pico PLUS Chemiluminescent Substrate (ThermoFisher Scientific) was used to detect the antibodies and the images were captured using a G:Box system (Syngene). Western blot images were quantified by densitometric analysis using ImageJ (National Institutes of Health).

2.5. Statistics

Data are presented as means ± standard deviation (SD) of the mean and were analyzed using GraphPad Prism software. Statistical comparisons were made using *t*-test, one- or two-way ANOVA with Bonferroni post hoc test as appropriate. In all tests, $p < 0.05$ was considered significant.

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Conflict of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.dib.2020.105290.

References

[1] E.E. Soltis, L.A. Cassis, Influence of perivascular adipose tissue on rat aortic smooth muscle responsiveness, Clin. Exp. Hypertens. A. 13 (1991) 277–296, https://doi.org/10.3109/10641969109042063.
[2] M. Gollasch, Vasodilator signals from perivascular adipose tissue, Br. J. Pharmacol. 165 (2012) 633–642, https://doi.org/10.1111/j.1476-5381.2011.01430.x.
[3] E. DeVallance, K.W. Branyan, K. Lemaster, I.M. Olfert, D.M. Smith, E.E. Pistilli, J.C. Frisbee, P.D. Chantler, Aortic dysfunction in metabolic syndrome mediated by perivascular adipose tissue TNFα- and NOX2-dependent pathway, Exp. Physiol. 103 (2018) 590–603, https://doi.org/10.1113/EP086818.
[4] S. Hildebrand, J. Stümer, A. Pfeifer, PVAT and its relation to brown, beige, and white adipose tissue in development and function, Front. Physiol. 9 (2018) 70, https://doi.org/10.3389/fphys.2018.00070.
[5] C.S. Ceron, G.T. do Vale, J.A. Simplicio, P. Passaglia, S.T. Ricci, C.R. Tirapelli, Data on the effects of losartan on protein expression, vascular reactivity and antioxidant capacity in the aorta of ethanol-treated rats, Data Br. 11 (2017) 111–116, https://doi.org/10.1016/j.dib.2017.01.006.
[6] S. Asano, G.C. O'Connell, K.C. Lemaster, E.R. DeVallance, K.W. Branyan, J.W. Simpkins, J.C. Frisbee, T.L. Barr, P.D. Chantler, Circulating leucocytes perpetuate stroke-induced aortic dysfunction, Exp. Physiol. 102 (2017) 1321–1331, https://doi.org/10.1113/EP086510.
[7] M.H.-H. Lee, S.-J. Chen, C.-M. Tsao, C.-C. Wu, Perivascular adipose tissue inhibits endothelial function of rat aortas via caveolin-1, PloS One 9 (2014), e99947, https://doi.org/10.1371/journal.pone.0099947.
[8] G.A. Payne, L. Borbouse, S. Kumar, Z. Neeb, M. Alloosh, M. Sturek, J.D. Tune, Epicardial perivascular adipose-derived leptin exacerbates coronary endothelial dysfunction in metabolic syndrome via a protein kinase C-beta pathway, Arterioscler. Thromb. Vasc. Biol. 30 (2010) 1711–1717, https://doi.org/10.1161/ATVBAHA.110.210070.