Data Article

Data on the expression and role of TREM-1 in the development of in-stent restenosis

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\textbf{A B S T R A C T}

The data presented in this article are related to the research article entitled “Increased serum TREM-1 level is associated with in-stent restenosis, and activation of TREM-1 promotes inflammation, proliferation and migration in vascular smooth muscle cells” (Wang et al., 2017) \cite{1}, which demonstrated that TREM-1 is expressed on vascular smooth cells (VSMCs) and promotes inflammation, proliferation and migration in cultured VSMCs. In this dataset, the expression of TREM-1 in leukocytes and endothelial cells of carotid artery after ligation was evaluated. The effect of TREM-1 on stenosis was analyzed in cultured human saphenous veins (HSVs) that spontaneously undergo remodeling which involves VSMC proliferation and migration.

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## Specifications Table

| Subject area       | Biology                          |
|--------------------|----------------------------------|
| More specific      | Vascular biology                 |
| subject area       |                                  |
| Type of data       | Graph, figure                    |
| How data was       | Immunofluorescence and tissue culture |
| acquired           |                                  |
| Data format        | Raw, analyzed                    |
| Experimental       | Expression pattern of TREM-1 in the ligated carotid artery was detected and the thickness of cultured human saphenous vein was measured after exposure to a TREM-1-specific antagonist (LP17) or agonist (monoclonal activating antibody) |
| factors            |                                  |
| Experimental       | Both the expression and molecular function of TREM-1 in the model of vascular stenosis was analyzed |
| features           |                                  |
| Data accessibility | The data are available with this article |

## Value of the data

- The data analyze changes in the expression of TREM-1 during the development of vascular stenosis.
- The data delineate the cellular source of TREM-1 in the vascular wall under basal conditions and after stenosis.
- The data provide evidence for the participation of TREM-1 signaling in the stenotic process.

## 1. Data

Dual immunofluorescence investigated whether TREM-1 was co-localized with endothelial cells (CD31<sup>+</sup>) or infiltrated leukocytes (CD45<sup>+</sup>) besides smooth muscle cells [1] within the vessel wall of the ligated carotid arteries (Fig. 1). Thickness of the intimal, medial and adventitial layers of the HSVs was measured after cultured ex vivo for 7 days with or without LP17 or TREM-1-activating antibody (Fig. 2).

## 2. Experimental design, materials and methods

### 2.1. Animals and carotid ligation

All animal experiments were conducted in accordance with experimental protocols approved by the Committee on Animal Resources of Shanghai Jiaotong University. Wild type C57BL/6 mice were obtained from Beijing Vital River Laboratory Animal Technology Co., Ltd (Beijing, China). Complete carotid ligation was performed on C57BL/6 mice anesthetized using 2.0% isoflurane, placed on a heated surgical board. A midline cervical incision was made and the left common carotid was isolated and ligated.

### 2.2. Immunofluorescence

Immunofluorescence was performed as previously described [2]. Paraffin-embedded sections of the carotid artery were deparaffinized and incubated with primary goat anti-TREM-1 antibody (#sc-
19312, Santa Cruz Biotechnology, Dallas, TX, USA), rabbit anti-PECAM-1 antibody (#sc-1506R, Santa Cruz Biotechnology, Dallas, TX, USA), or rabbit anti-CD45 (#ab23910, Abcam, Cambridge, UK). After incubation with the appropriate fluorescence-conjugated secondary antibodies (1:1000 dilution, Alex

Fig. 1. Infiltrated leukocytes but not endothelial cells express TREM-1 in the ligated carotid artery. Left common carotid arteries of C57BL/6 mice were isolated 21 days after ligation. Expression of TREM-1 in the injured vessels was determined by immunofluorescence (IF) analysis. Endothelial cells and infiltrated leukocytes were detected by staining of CD31 and CD45, respectively. Merge shows evident co-localization of TREM-1 with CD45 but not with CD31. Shown are typical images from 4 independent experiments.

Fig. 2. Medial thickening of the cultured human saphenous vein is modulated by TREM-1 signaling. Human saphenous vein (HSV) explants were subjected to ex vivo culture for 7 days with or without a synthesized human TREM-1 inhibitory peptide (hLP17) or a monoclonal TREM-1-specific activating antibody (Ab). (A) Shown are representative images of HSV sections with immunostaining for α-smooth muscle actin (brown). (B) Quantification of intimal, medial and adventitial thickness. Data are expressed as mean ± SD of 4 independent experiments. *p < 0.05, **p < 0.01 versus control.
Fluor 546 and 488, respectively; Invitrogen, Carlsbad, CA, USA), images were acquired using an inverted epi-fluorescence microscope (Olympus BX61) equipped with a DP72 camera.

2.3. Human saphenous veins culture ex vivo

HSV, not required for surgery, were collected from discards after coronary artery bypass graft surgery and were cultured as previously described [3]. Briefly, the vein segments were opened longitudinally and cultured individually with luminal surface facing up in 12-well plates in RPMI 1640 medium supplemented with 30% FBS, 2 mmol/l L-glutamine, 100 IU/ml penicillin and 100 μg/ml streptomycin for 7 days with or without LP17 or TREM-1-activating antibody. The vein segments were then fixed and embedded in paraffin. Cross-sections were cut and stained with α-smooth muscle actin. Thickness of intima, media, and adventitia was measured by Image-Pro Plus 6.0.

2.4. Statistics

Data are expressed as mean ± SD or SEM from 4 to 7 independent experiments. Differences between two groups were analyzed by two-tailed Student’s t-test. Probability values less than 0.05 were considered statistically significant. All statistical analysis was performed with SPSS 19.0 for Windows (SPSS, Inc., Chicago, IL, USA).

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Transparency document. Supporting information

Transparency data associated with this article can be found in the online version at https://doi.org/10.1016/j.dib.2017.11.065.

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