Supporting Information

Attenuating endothelial leakiness with self-assembled DNA nanostructures for pulmonary arterial hypertension

Qian Liu,[‡ab] Di Wu,[‡a] Binfeng He,[a] Xiaotong Ding,[a] Yu Xu,[a] Ying Wang,[c] Mingzhou Zhang,[a] Hang Qian,*[a] David Tai Leong,*[d] Guansong Wang,*[a]

a Institute of Respiratory Diseases, Xinqiao Hospital, Third Military Medical University, Chongqing 400037, China.

b Laboratory of Pharmacy and Chemistry, and Laboratory of Tissue and Cell Biology, Lab Teaching & Management Center, Chongqing Medical University, Chongqing 400016, China.

c Department of Cardiology, Xinqiao Hospital, Third Military Medical University, Chongqing 400037, China.

d Department of Chemical and Biomolecular Engineering, National University of Singapore, Singapore 117585, Singapore.

‡These authors contributed equally to this work.

* Corresponding authors.

E-mail addresses: hqian@tmmu.edu.cn (H. Qian), cheltwd@nus.edu.sg (D. T. Leong), wanggs@tmmu.edu.cn (G. Wang)

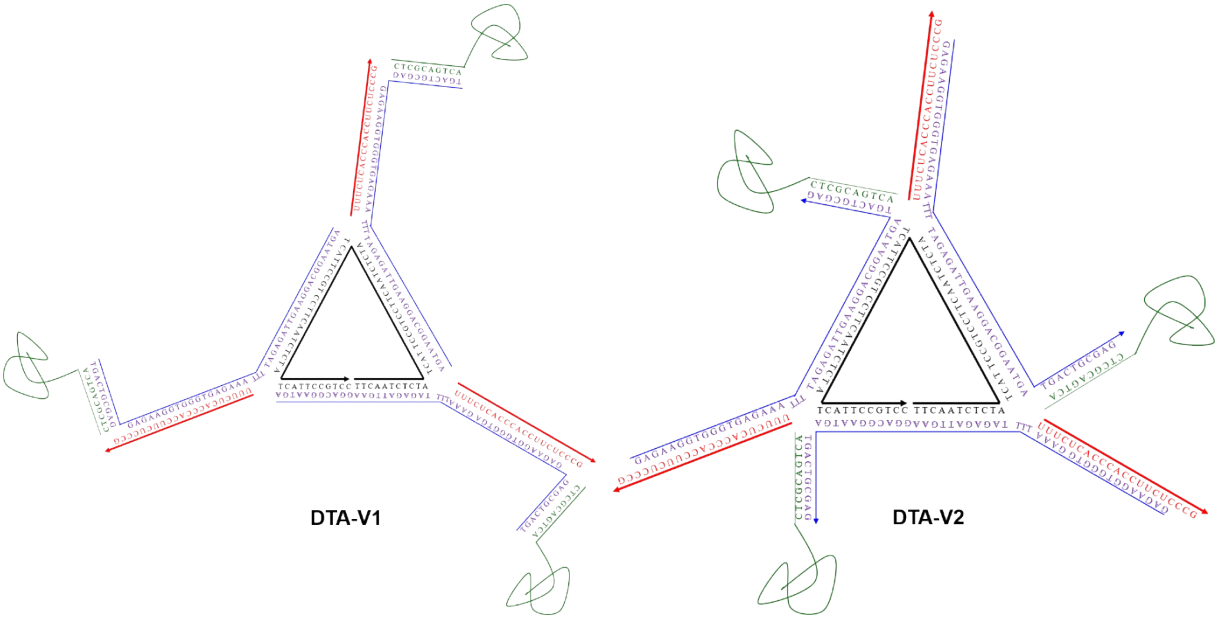


Figure S1. Base pairing design of DTA-V1 and DTA-V2.

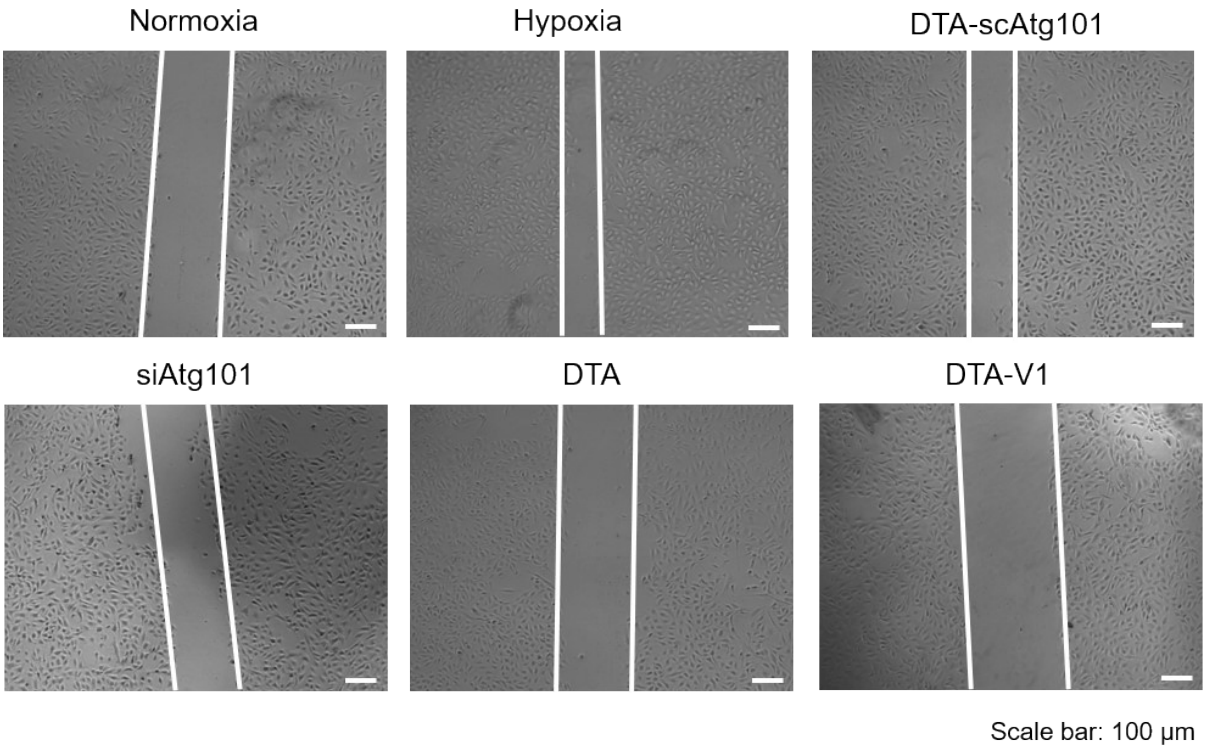
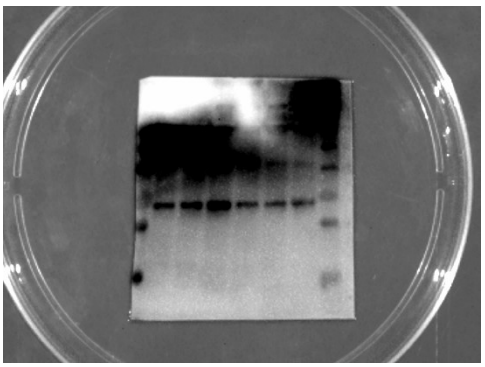
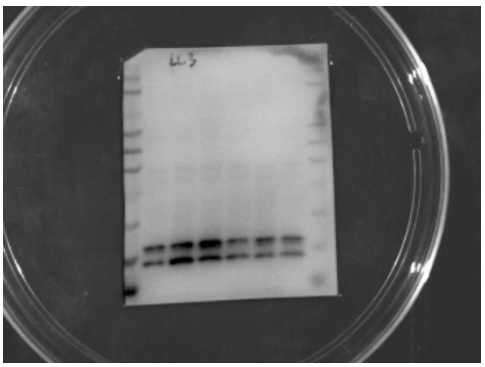


Figure S2. Scratch assay showed that DTA-V1 effectively inhibited the proliferation of HPAECs. Cells were treated with DTA-V1 for 24 h. siAtg101 concentrations was 600 nM.

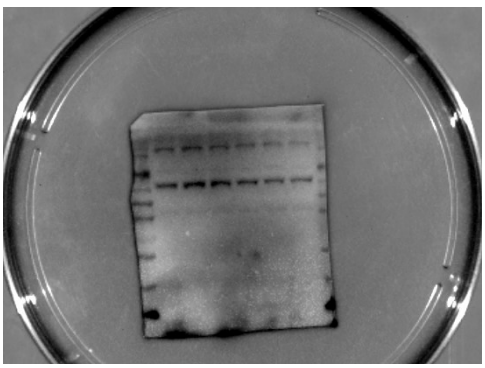
Atg101



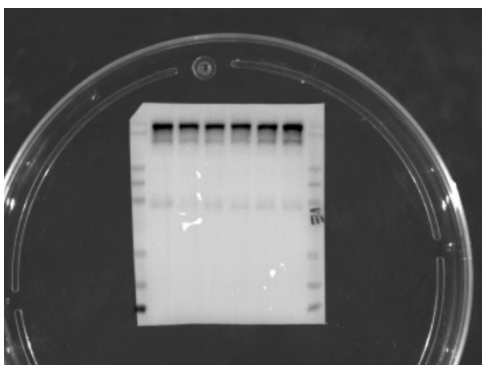
LC3



Beclin-1



VE-Cadherin



β-actin

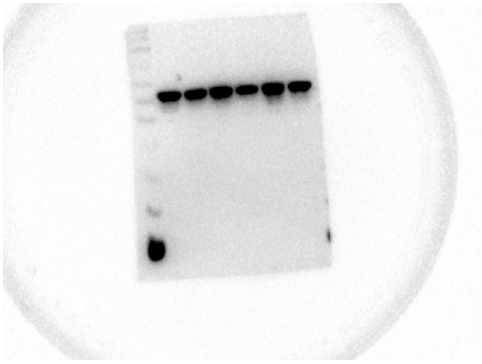


Figure S3. Uncropped data for the western blots in Fig. 4B.

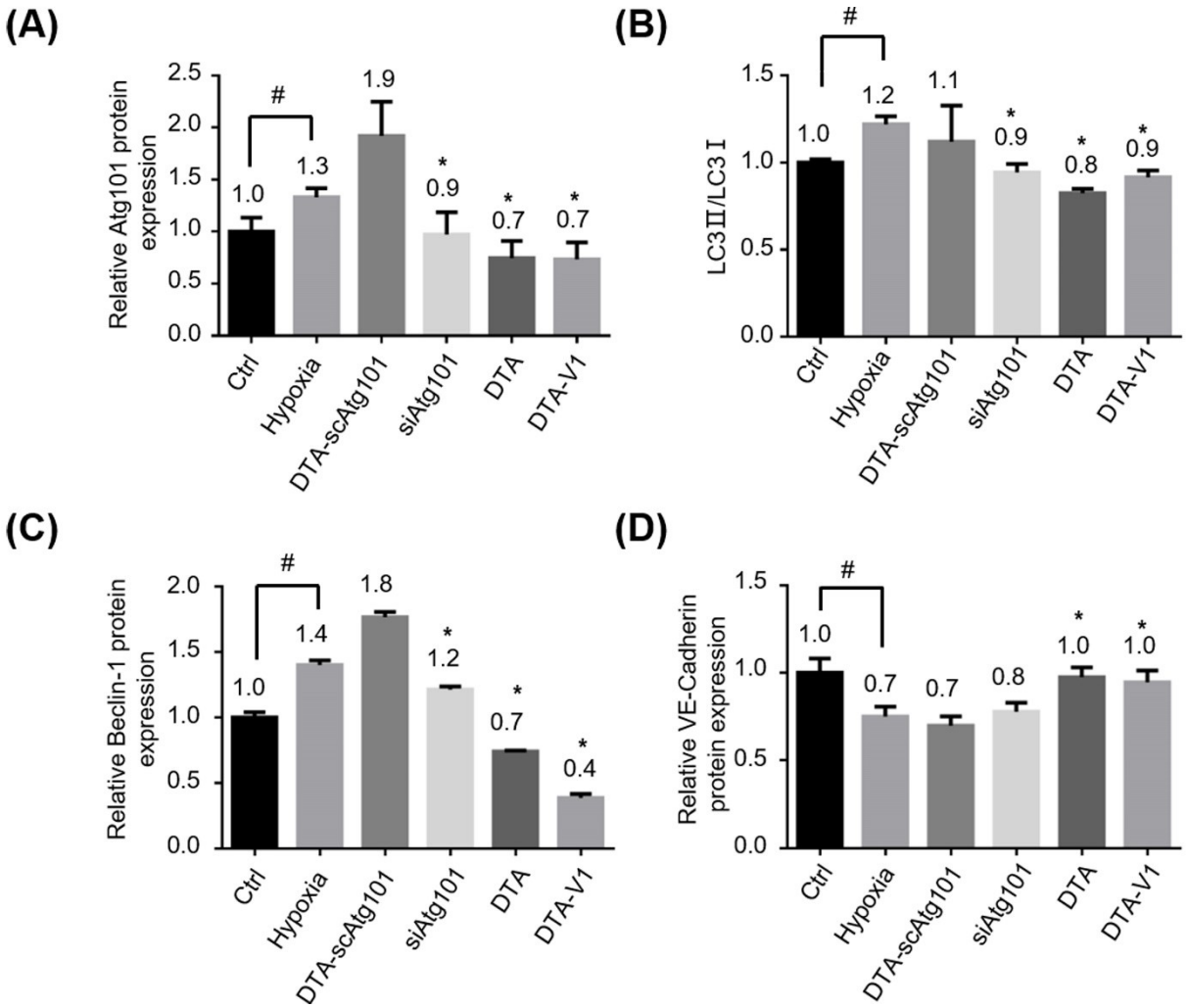


Figure S4. Semi-quantitative analysis of the protein expressions in Figure 5B. (A) Atg101; (B) LC3; (C) Beclin-1; (D) VE-Cadherin. All the data of figures represent the mean value ± SD (n = 3). p-values were calculated using one-way ANOVA, *$p< 0.05$ compared with hypoxia group, $^{\#}p<0.05$ indicate connected groups.