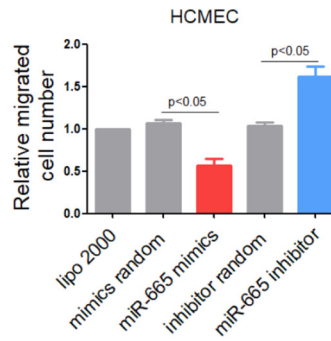
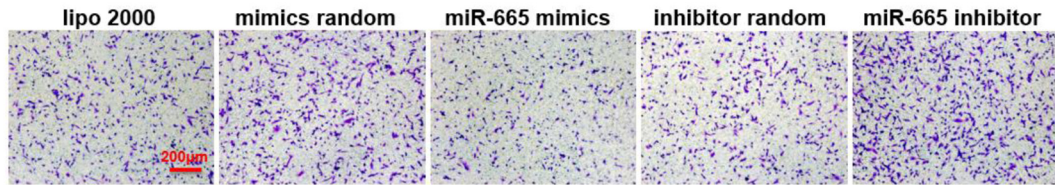
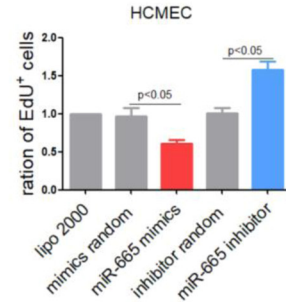
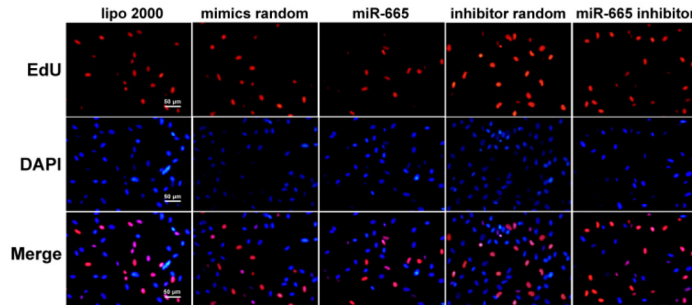


SUPPLEMENTARY FIGURES

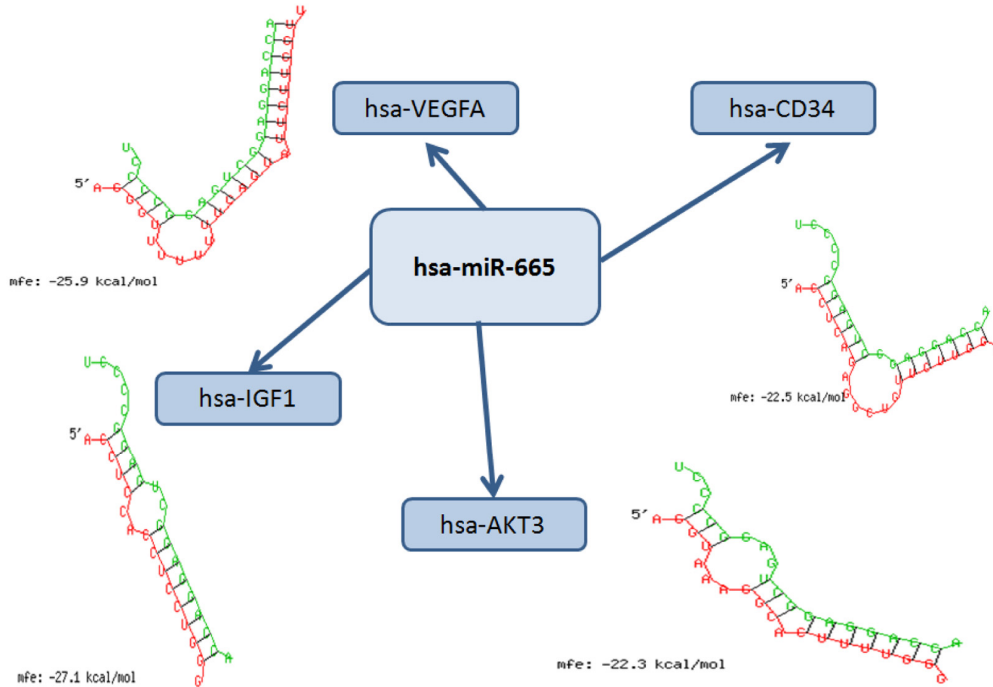
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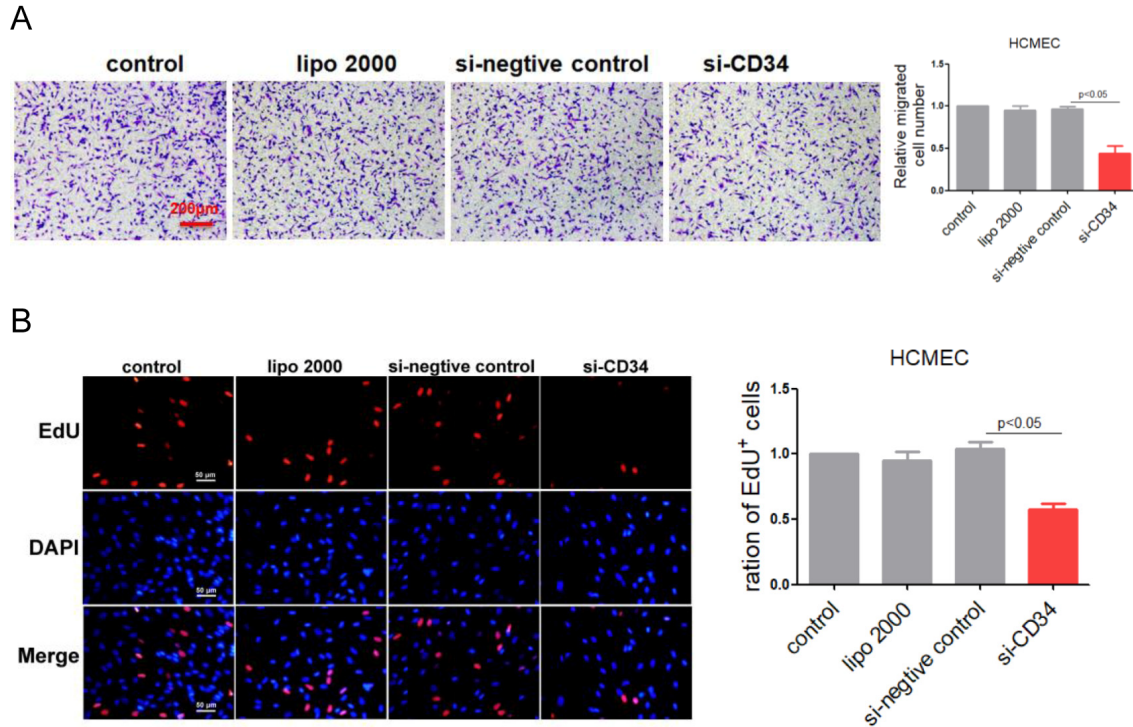
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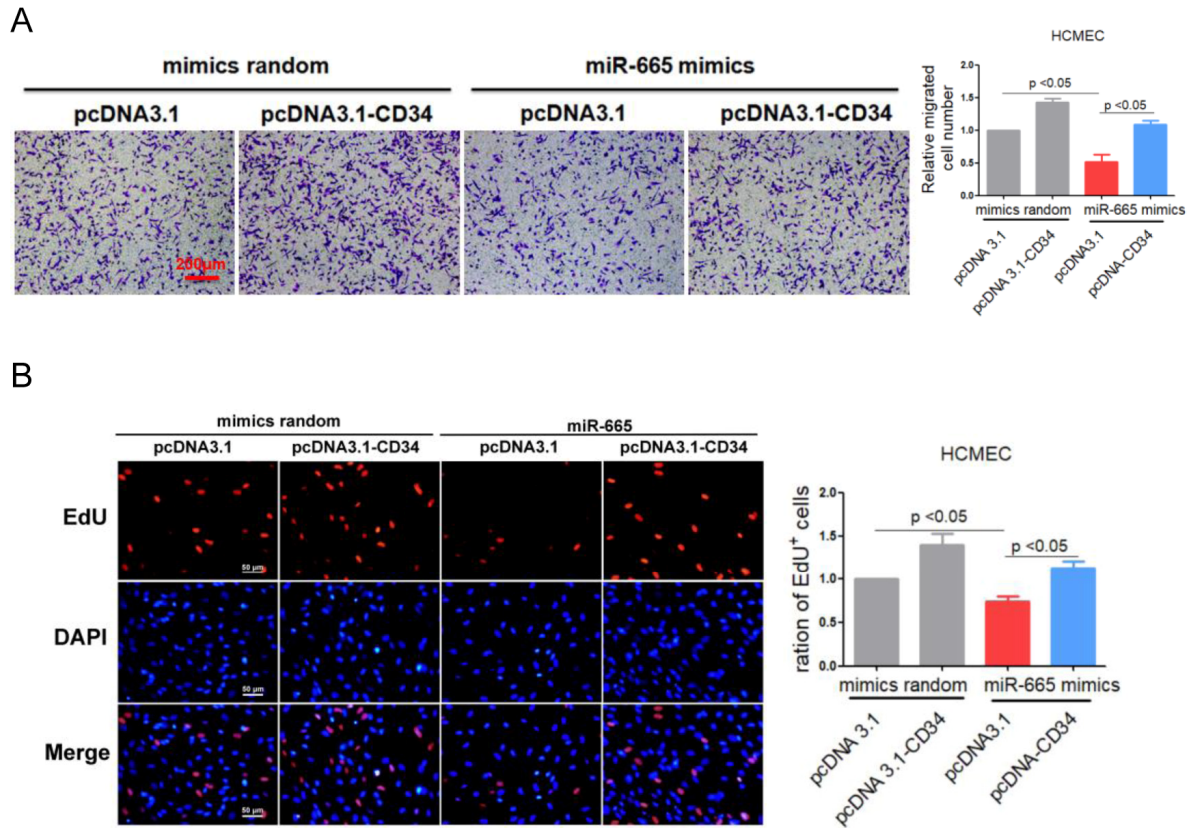
Supplementary Figure 1. MiR-665 impairs endothelium function in vitro. (A) Migration evaluated by transwell experiment in HCMEC cells. (B) Proliferation detected by EdU incorporation assays in HCMEC cells.



Supplementary Figure 2. Predicted targets of miR-665. The Bielefeld Bioinformatics Server was used to search for mRNA that could interact with miR-665. CD34, IGF1, VEGFA and AKT3 were suggested as targets of miR-665.

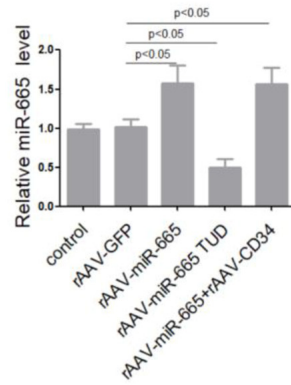


Supplementary Figure 3. Down-regulation of CD34 impairs endothelium function in vitro. (A) Migration valuated by transwell experiment in HCMEC cells. (B) Proliferation detected by EdU incorporation assays in HCMEC cells.

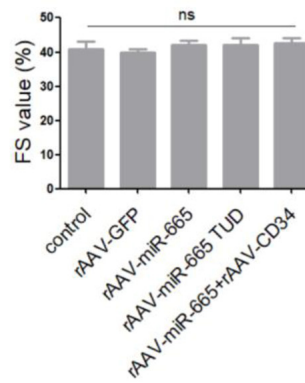
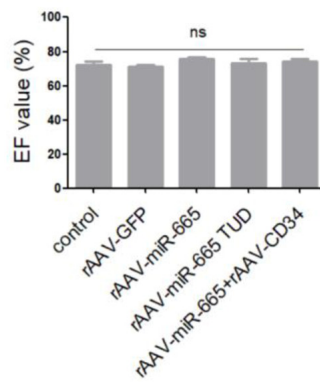


Supplementary Figure 4. Re-expression of CD34 reverses miR-665 induced endothelium dysfunction in vitro. (A) Migration evaluated by transwell experiment in HCMEC cells. (B) Proliferation detected by EdU incorporation assays in HCMEC cells.

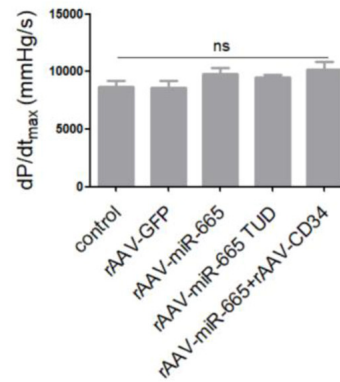
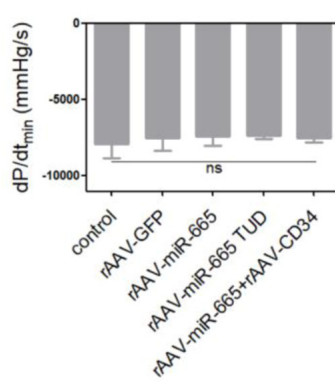
A



B



C



Supplementary Figure 5. MiR-665 does not affect heart function under normal conditions. (A) Relative cardiac expression of miR-665 detected by real-time PCR. **(B)** Echocardiographic detection in treated mice. **(C)** Hemodynamic analysis measured by Millar cardiac catheter system in treated mice.