



Ein Unternehmen der
Johann Wolfgang Goethe-Universität
Frankfurt am Main

Benefits

- New target for treating age-related heart disease: ZBTB16 as a key regulator of endothelial and cardiac aging.
- Potential to intervene early in the disease process rather than only addressing downstream effects.
- Promising commercial potential due to age-related heart dysfunctions affecting many patients.

Project Status

- A proof-of-concept has been demonstrated in vitro and in aged mouse models in vivo, including beneficial effects of ZBTB16 overexpression to preserve cardiac function in the aging heart.
- First exploratory experiments indicate that ZBTB16 expression may also be pharmacologically inducible.

Patents and Publications

- [WO2025093713A1](#)
ZBTB16 AS A FACTOR FOR CARDIAC AGING AND DISEASE
- Stolz et al. (2026) Endothelial ZBTB16: a molecular shield against cardiac aging. [European Heart Journal](#). 47:1186-1198.

Offer

- Co-development or strategic partnership
- Technology can be licensed or assigned
- Collaborations for further development welcome

INNOVECTIS
Gesellschaft für Innovations-
Dienstleistungen mbH
Altenhöferallee 5
60438 Frankfurt am Main, Germany
Tel: +49 69 25 61 632-0
christoph.emmerich@innovectis.de



Technology Offer

ZBTB16 as a key factor for cardiac aging and disease

Background:

Cardiovascular aging is a major driver of heart dysfunction, particularly diastolic dysfunction and fibrotic remodeling, while endothelial cell impairment is increasingly recognized as a central contributor to this process.

Scientists from the Goethe University Frankfurt, in particular from the Institute of Cardiovascular Regeneration, have identified **ZBTB16 as a master regulator of endothelial integrity and vascular niche homeostasis in the aging heart.**

In vitro and in vivo studies showed that repression of ZBTB16:

- induces endothelial senescence,
- impairs angiogenic function,
- promotes pro-fibrotic paracrine signaling,
- causes premature cardiac aging phenotypes including diastolic dysfunction

Importantly, endothelial overexpression of ZBTB16 reverses these effects and improves cardiac function in aged mice.

Mechanistically, the studies identified nuclear receptor interacting protein 1 (NRIP1) as a downstream target suppressed by ZBTB16, thereby limiting fibroblast activation and pro-fibrotic signaling.

Furthermore, it was also shown that ZBTB16-deficient endothelial supernatants activate fibroblasts, induce cardiomyocyte hypertrophy, and impair neuronal sprouting, highlighting a broad role for ZBTB16 in preserving cardiac microenvironmental crosstalk.

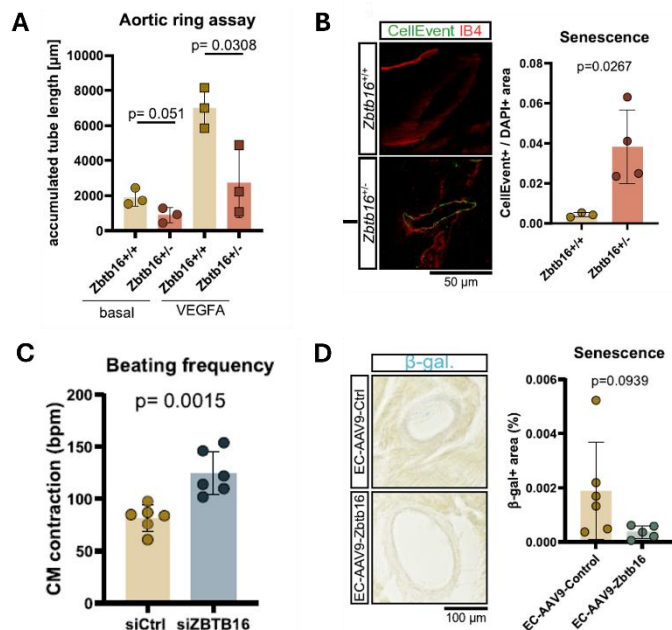


Figure 1: ZBTB16 as a promising target for interventions aimed at preventing age-related cardiac dysfunction. (A) Ex vivo aortic ring assay of Zbtb16^{+/+} and Zbtb16^{+/-} mice. (B) Senescence-associated β-galactosidase staining (CellEvent) of Zbtb16^{+/+} and Zbtb16^{+/-} mouse hearts. (C) siCtrl or siZBTB16-transfected HUVEC were directly co-cultured with neonatal rat cardiomyocytes and cardiomyocyte beating frequency is shown. (D) Overexpression of Zbtb16 in endothelial cells by targeted AAV9 vectors reduces senescence and improves cardiac function in 20-month-old mice.