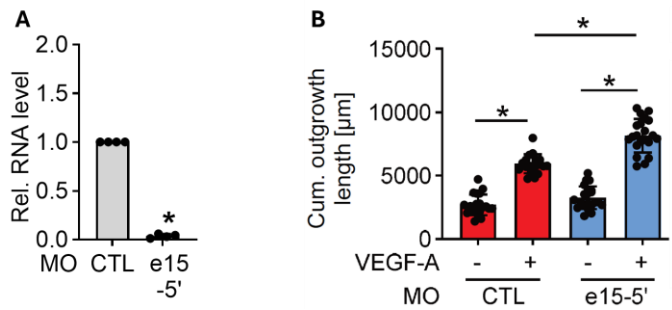
Technology Offer

New Morpholino for the Treatment of Cardiovascular Diseases and Preeclampsia

Researchers at Goethe University Frankfurt have developed a new splice-blocking morpholino oligonucleotide that selectively reduces sFLT1, a key anti-angiogenic factor and biomarker in several cardiovascular diseases and preeclampsia. The morpholino binds to a defined splice site in the FLT1 pre-mRNA and prevents inclusion of the alternative exon that generates the soluble isoform (sFLT1), while preserving membrane-bound FLT1 and thus physiological pro-angiogenic signaling.

Key findings include:

- **High potency:** The morpholino potently reduces >95% sFLT1 mRNA after 24h and markedly reduces sFLT1 protein levels in primary human umbilical vein endothelial cells (HUVECs).
- **Isoform selectivity:** Membrane-bound FLT1 remains unchanged.
- **Potential to restore endothelial function:** VEGF-A-mediated sprout length and sprout number increase after morpholino-mediated sFLT1 knockdown, demonstrating enhanced angiogenic capacity.



The splice-blocking morpholino e15-5' reduces sFLT1 production and enhances angiogenic capacity. (A) RNA level of sFLT1 measured by RT-qPCR after morpholino treatment (10 µM, 24h; n=4). **(B)** Spheroid outgrowth assay of HUVECs electroporated with e15-5' and stimulated with VEGF-A (30 ng/mL, 16 h); cumulative sprout length quantified (n = 21). One-way ANOVA with Tukey's post-hoc test.

The clinical need is substantial: sFLT1 is elevated in multiple forms of heart failure and cardiomyopathy, where anti-angiogenic signaling contributes to endothelial dysfunction and disease progression. Heart failure (systolic and diastolic) affects around 55 million patients worldwide, and the global heart-failure drug market is projected to reach 33.7 billion USD by 2032, underscoring the demand for novel, mechanism-based therapies. This morpholino offers a unique, isoform specific approach that is not addressed by current standard therapies so far.

Beyond heart failure, elevated sFLT1 is a key driver of preeclampsia, providing an additional, clearly defined indication and broadening the therapeutic and commercial potential of this targeted morpholino platform.

The Frankfurt University team looks forward to cooperating with commercial partners to transfer the invention towards industry and market exploitation.

Benefits

Novel therapy option for several cardiovascular diseases as well as preeclampsia

Project Status

- **New splice-blocking morpholino, which selectively targets sFLT1 and thereby preserves physiological pro-angiogenic signaling**
- **Development of a mouse model to test the morpholino *in vivo***

Patents and Publications

- **EP patent application EP25218896.6: filed on 27. November 2025 by Johann Wolfgang Goethe-University Frankfurt**
- **Lam et al., 2026, LINC00607 facilitates endothelial VEGF-A receptor FLT1 splicing**

Offer

- **Technology can be licensed or assigned**
- **Collaborations for further development welcome**

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