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Background

- Vascular instabilities resulting from dysautonomia such as postural orthostatic tachycardia syndrome (POTS) remain poorly understood, especially in the context of ME.
- A pan epigenomic analysis revealed miR-181a-5p as one of a key player in ME+POTS comorbidity, related to clinical feature of vascular instability.

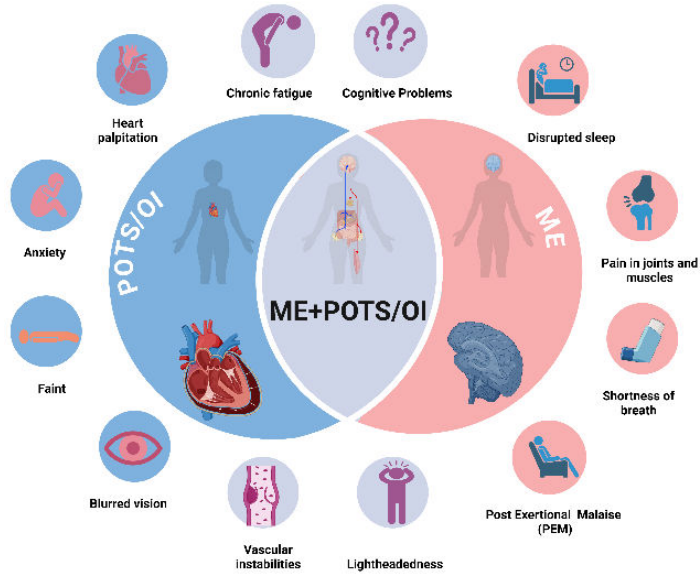


Figure 1. Distinct and shared clinical features of ME and POTS

Hypothesis and objective

- Dysregulated miR-181a-5p in ME+POTS may alter calcium-handling related genes leading to autonomic dysfunction.
- The aim is to characterize the molecular and functional effects of miR-181a-5p on cardiomyocyte calcium dynamics.

Methods

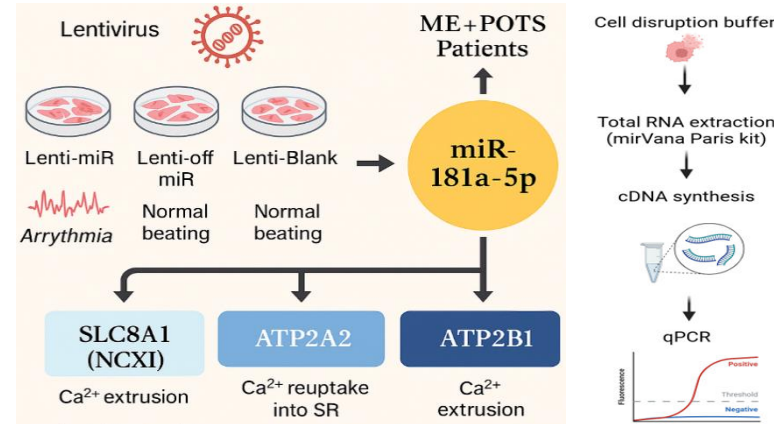


Figure 2. Methodology of the study

Results

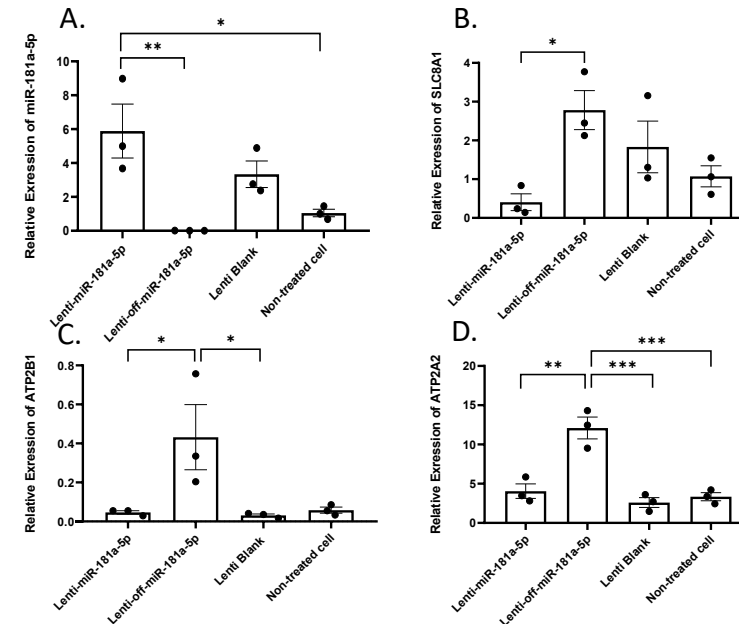


Figure 3. A. Relative expression of miR-181a-5p in different treatments. B,C,D. Relative expression of SLC8A1, ATP2B1, and ATP2A2 in different treatments.

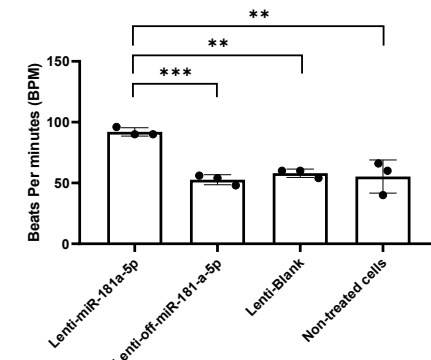


Figure 4. Beating rate of hiPSC-derived cardiomyocytes under different lentiviral treatments.

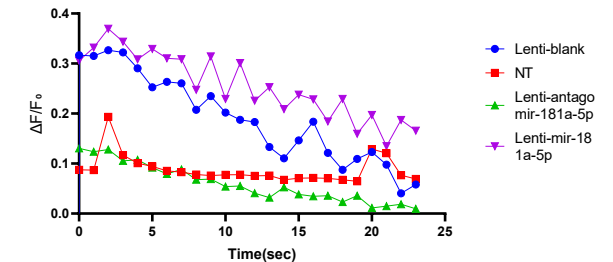


Figure 5. Mean fluorescence intensity of calcium transients over time across different treatment groups

Discussion

By targeting genes such as *SLC8A1* (*NCX1*), *ATP2A2* (*SERCA2*), and *ATP2B1* (*PMCA1*), miRNAs like *miR-181a-5p* may alter intracellular calcium dynamics, leading to impaired cardiomyocyte function and aberrant autonomic signaling. These findings emphasize the potential of miRNA-based biomarkers and their therapeutic modulation as a promising prospect.

Acknowledgment

A special thank to all ME patients and participants for their contribution to this study.