DOI: doi.org/10.47363/JCRRR/CardioCare2026/2026(7)2

# CardioCare 2026: Innovations in Cardiovascular Health and Treatment

Conference Proceedings May 25, 2026 - United Kingdom

## Micro RNA-based Regulation of the Sinoatrial Node: A Look into Pacemaking Future?

Dr. Hanan Nur

North Manchester General Hospital, Manchester University NHS Foundation Trust, UK

**Abstract**

The sinoatrial node (SAN) constitutes the dominant pacemaker of the heart, generating spontaneous electrical activity that governs cardiac rhythm and ensures effective myocardial contraction. Anatomically, the SAN is composed of a heterogeneous population of specialised pacemaker cells located at the junction of the superior vena cava and the right atrium, supported by a paranodal region that contributes to impulse propagation, rhythm stability, and may serve as a secondary pacemaker during primary pacemaker dysfunction. At the molecular level, SAN automaticity is critically dependent on hyperpolarisation-activated cyclic nucleotide-gated channel 4 (HCN4), which mediates the pacemaker current (If) and regulates the slope of diastolic depolarisation. Increasing evidence implicates microRNAs as pivotal post-transcriptional regulators of cardiac electrophysiology, with emerging roles in both physiological and pathological modulation of heart rate. In particular, microRNA-486-3p has been identified as a regulator of HCN4 mRNA expression, suggesting a novel mechanism for modulating SAN pacemaker activity at the transcriptional level. This review synthesises current knowledge regarding the structural and molecular regulation of the SAN, with emphasis on the functional interaction between HCN4 and miR-486-3p, and critically evaluates the therapeutic potential of microRNA-based strategies for the management of sinus tachycardia. By comparing microRNA-mediated modulation of HCN4 expression with established pharmacological interventions such as ivabradine, which exerts its effects through direct channel blockade, this review explores the translational implications of targeting pacemaker regulation upstream at the gene expression level.

Elucidation of these mechanisms may facilitate the development of more selective and potentially safer therapeutic approaches for heart rate control, particularly in clinical contexts where conventional treatments are limited or contraindicated.