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CRISPR/Cas9 ablation of individual miRNAs from a miRNA family reveals their individual efficacies for regulating cardiac differentiation

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MicroRNAs are quickly entering the causal fray of developmental defects. These small, non-coding RNAs use a 7-8 base-pair seed sequence to target a corresponding sequence on one or multiple mRNAs resulting in rapid down-regulation of translation. miRNAs can also control protein amounts in cells. As a result, if miRNAs are over or under expressed during development protein homeostasis can be compromised resulting in defects in the development of organ systems. Here, we show that during differentiation of embryonic stem cells, individual miRNAs that reside in the miRNA17 family (composed of 15 miRNAs) do not share the same function even though they have the same seed sequence. The advent of CRISPR/CAS9 technology has not only yielded a true observation of individual miRNA function, it has also reconnected advanced molecular biology approaches to classical cell biology approaches such as gene rescue. We show that miRNA106a and to a lesser extent miR17 and 93 target the cardiac suppressor gene Fog2, which specifically suppress Gata-4 and Coup-TF2. However, when each miRNA is knocked out, we find that their targeting efficacies for Fog2 differ resulting in varying degrees of cardiac differentiation.

Biography

G Ian Gallicano has completed his PhD in 1994 from Arizona State University afterwhich he has completed his Post-doctoral studies at The University of Chicago/ Howard Hughes Institute. He has been the Director of the Transgenic Shared Resource at the Lombardi Comprehensive Cancer Center and is currently implementing CRISPR gene editing technology at GUMC. He has published more than 60 papers in reputed journals and is currently an Editorial Board Member for the journal *Stem Cells* as well as the Editor-in-Chief for *American Journal of Stem Cells*.

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