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Loss of miR-10b triggers depletion of interstitial cells of Cajal in diabetic mice

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Both acute and chronic hyperglycemias in diabetes are linked to gastrointestinal (GI) complications that can change normal function of motility in the gastrointestinal (GI) tract. The most common pathological abnormality in diabetic patients in the GI tract is characterized by depletion of interstitial cells of Cajal (ICCs). ICCs are pacemakers in the GI tract that regulate GI motility through spontaneous electrical slow waves. ICCs exclusively express the receptor tyrosine kinase KIT, which is required for the normal development and maintenance of ICCs. However, underlying molecular mechanisms in depletion of ICCs in diabetic patients are largely elusive. Here we report a microRNA mediated ICC depletion in the diabetic animal model. We identified miR-10b-5p as a diabetic cell marker that was dramatically diminished in the diabetic ICCs. MiR-10b-5p targets an epigenetic repressor, nuclear receptor co-repressor 2 (NCOR2), which negatively regulates expression of KIT. Expression of KIT and NCOR2 proteins was negatively related in diabetic intestine where KIT+ ICCs were found in lower concentration. ICC restricted miR-10b knockout in mice resulted in depletion of ICCs in the intestine. Our findings on KIT regulation by miR-10b-5p targeted NCOR2 offer a new insight into how ICCs is phenotypically changed and become non functional in diabetic conditions. Moreover, this study suggests miR-10b-5p may be potentially an attractive therapeutic target to restore KIT expression in diabetic ICCs of patients with GI dysmotility.

Biography

Seungil Ro has obtained his PhD in Cell and Molecular Biology from the University of Nevada, Reno, USA in 2002, where he has been Associate Professor since 2015 in the Department of Physiology and Cell Biology in School of Medicine. His research interest includes the roles of microRNAs that regulate gastrointestinal smooth muscle motility and epigenetic remodeling. He has 35 papers published to his credit.

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